Out of Sight, Out of Mind

Population Estimates and Dynamic Interplay between Age-related Decline in Hearing and Cognitive Functioning during Late Life.

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A thesis submitted for the degree of

Doctor of Philosophy of

The Australian National University.

January 2013

Declaration

Except where otherwise acknowledged, this thesis is my own original work and has not been submitted for a higher degree at any other university or institution. I developed the research questions, harmonized and pooled the data, and conducted and interpreted all statistical analyses presented herein. This thesis draws on secondary data analysis of the Dynamic Analyses to Optimise Ageing (DYNOPTA) project, a collaborative program that has pooled nine longitudinal studies of ageing. Within DYNOPTA I advised on data pooling strategies, performed harmonization and was responsible for data management.

Kim Matthew Kiely

"I must live almost alone, like one who has been banished; I can mix with society only as much as true necessity demands. If I approach near to people a hot terror seizes upon me, and I fear being exposed..."

Ludwig Van Beethoven

Heiligenstadt

6 October 1802

Acknowledgment

First and foremost, I must express my sincere gratitude to my supervisor Professor Kaarin Anstey for guiding me throughout my PhD candidature and always offering encouragement, honest feedback and sound advice. I especially would like to thank Kaarin for providing excellent research training, and presenting opportunities to learn from, and collaborate with, colleagues based locally and abroad.

I would also like to acknowledge my panel advisors to whom I am greatly indebted: Professor Mary Luszcz for the opportunity to work with ALSA and always finding the time to read my work (even when on LSL); Dr Denis Gerstorf for his attention to detail, meticulous advice on statistical methodology and being a gracious host during my visit to Penn State in 2009; And Dr Lesley Ross for her mentoring, GSA drinky-poos and grits!

Thank you to all other DYNOPTA collaborators, in particular Professor Paul Mitchell and Dr Bamani Gopinath for their sharing their expertise, commenting on my manuscripts and providing the opportunity to work with the Blue Mountains Eye Study.

Finally, I must give special thanks to Dr Richard Burns for the coffee, Sunny and Bindi for their energy and eternal optimism, Dylan for giving me a new cause for sleepless nights - And of course, to Emma for her support, patience, and unwavering affection.

Précis

Age-related sensory loss and major neurocognitive impairment are two of the leading drivers of non-fatal disability burden among the oldest-old, and are often reported to co-occur. Both biological and social explanations have been given to account for links between these two functional domains. This thesis explores inter-associations between age-related hearing-loss with cognitive function. The broad substantive aims of this dissertation are: 1) to document the levels of hearing impairment, dual sensory loss, and co-morbid hearing-loss with cognitive impairment in an older adult population; 2) to identify predictors of decline in hearing acuity and its association with all-cause mortality risk; 3) to investigate longitudinal pathways between hearing thresholds, hearing aid use and processing speed.

The Dynamic Analyses to Optimise Ageing (DYNOPTA) project is a collaborative inter-disciplinary project that has pooled nine Australian longitudinal studies of ageing. The DYNOPTA project constitutes an important methodological backdrop to this thesis. Data pooling is advantageous because it can enhance representativeness of a population, increased statistical power and allows for direct replication of effects. However, variability in study protocols and the need to orientate functionally equivalent measures onto a common scale can create analytic challenges. A subsidiary aim of this thesis will be to illustrate and evaluate the use of harmonised longitudinal data pooled from independently designed epidemiological surveys.

This research presented in this thesis primarily draws upon data from two contributing DYNOPTA studies that began in the early 1990s and are ongoing. These two studies were selected because they collected functionally equivalent clinical measures of hearing, vision and cognition, as well as a range of comparable contextual variables including data on socio-demographics, health, noise exposure, and hearing aid

use. Multistate Markov Chain models estimated transition rates and expected years lived with sensory impairment. Joint Survival-Growth Curve models demonstrated that hearing thresholds were associated with increased mortality risk in women but not in men. Linear Mixed Models were used to identify predictors of hearing trajectories. Bi-variate Dual Change Score models demonstrated that low levels of hearing were leading indicators of subsequent rates of decline in processing speed. Finally, hearing-aid use was shown to be associated with improved levels of processing speed after adjusting for the effects of hearing thresholds, but did not attenuate rates of decline in processing speed.

Hearing loss and cognitive impairment are highly prevalent and contribute to a significant number of years lived with functional impairment in late life. Links between hearing and cognition may be due to common biological processes. Alternatively, hearing loss could limit opportunities to engage in activities that promote and maintain cognitive reserves. Reductions in cognitive resources may also mean that older adults are less well equipped to deal with sensory ageing. In the context of this thesis, the main benefits of pooling and harmonization were the capacity to derive coarse population level estimates and the fostering of inter-disciplinary collaboration. However, it was necessary to return to the use of single study data to facilitate investigations into more fine grained causal pathways between hearing and cognition.

Publications Arising from this Thesis

Journal Articles

Kiely, K. M., Luszcz, M. A., Piguet, O., Christensen, H., Bennett, H., & Anstey, K. J. (2011). Functional equivalence of the National Adult Reading Test (NART) and Schonell reading tests and NART norms in the Dynamic Analyses to Optimise Ageing (DYNOPTA) project. Journal of Clinical and Experimental Neuropsychology, 33(4), 410-421.

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Kiely, K. M., Gopinath, B., Mitchell, P., Luszcz, M., & Anstey, K. J. (2012). Cognitive, Health, and Sociodemographic Predictors of Longitudinal Decline in Hearing Acuity Among Older Adults. The Journals of Gerontology Series A: Biological Sciences and Medical Sciences. doi: 10.1093/gerona/gls066

Conference Abstracts

Kiely, K.M., Luszcz, M., & Anstey, K. J. (2013). Do hearing aids protect against cognitive decline or does higher cognitive function predict hearing aid use? Paper presented at the International Association for Gerontology and Geriatrics 20th World Congress. Seoul, Korea.

Kiely, K. M., Cherbuin, N., Gerstorf, D., Luszcz, M., & Anstey, K. J. (2011). Lateralization and brain aging: a comparison of static and dynamic longitudinal associations between cognitive functioning and pure-tone thresholds in the left and right

ear. The Gerontologist, 51(supp2), 268. Paper presented at the The Gerontological Society of America's 64th Annual Scientific Meeting "Lifestyle→Lifespan". Boston,USA.

Kiely, K. M., Gopinath, B., Mitchell, P., Luszcz, M.A., & Anstey, K. J. (2011). Age-related hearing loss and its association with health and longevity in older adults. Evidence from DYNOPTA. Paper presented at the International Association for Gerontology and Geriatrics 9th Asia/Oceania Regional Congress. Melbourne, Australia.

Kiely, K. M., Gopinath, B., Mitchell, P., Luszcz, M.A., & Anstey, K. J. (2011). Intra-individual and inter-individual effects of cognitive impairment on decline in hearing during late life. Combined Abstracts of the 2011 Australian Psychology Conference, 198-199. Paper presented at the 46th Annual Australian Psychological Society Conference. Canberra, Australia.198-199

Kiely. K. M., Anstey. K, & The DYNOPTA Investigators. (2009). The DYNOPTA project and variable harmonisation: Establishing a common ground for combining nine Australian longitudinal studies of ageing. Australasian Journal on Ageing, 28(S2), A63. Paper presented at the Australian Association of Gerontology Annual Conference. Canberra, Australia.

Kiely, K. M., Anstey, K. J., & The DYNOPTA Investigators. (2009). Variable harmonisation in the DYNOPTA project: Establishing a common ground for combining sensory functioning data across nine Australian longitudinal studies of ageing. Abstracts and Proceedings (page 30, 4E). Paper presented at the 8th National Conference of Emerging Researchers in Ageing: Melbourne, Australia.

Colloquia, Committee and Community Presentations

Kiely, K. M. (September 2012). "Epidemiology of Age-related Hearing Loss, links with cognitive function and the role of hearing aids". Talk to the *ACT AAG Annual General Meeting*, Council of the Ageing (COTA), Canberra

Kiely, K. M. (June 2012). "Out of sight, out of mind: Age-related Hearing Loss and its Association with Cognitive Impairment". Talk to the *Centre for Research on Ageing Health and Wellbeing*, The Australian National University

Kiely, K. M. (March 2012). "Epidemiology of Age-related Hearing Loss and its Association with Cognitive Impairment". Talk at the *ACT Australian Psychological Society (APS) Ageing Interest Group (PAIG) Breakfast Meeting*

Kiely, K. M. (September 2011). "Late-life Associations between Cognitive and Auditory Functioning". Paper presented at the *CMHR Student Colloquium.* Centre for Mental Health Research, The Australian National University

Kiely, K. M. (October 2011). "PhD Update: Declines in Sensory and Cognitive Functioning in the Years Before Death". Paper presented at the *Annual DYNOPTA Retreat.* CMHR, The Australian National University.

Kiely, K. M. (March 2011). "The Epidemiology of Age-related Hearing Loss". Paper presented at the *3rd Australian Association of Gerontology Intergenerational Forum*. NATSEM, University of Canberra.

Kiely, K. M. (July 2010). "Does the Orientation of Time Alter Developmental Dynamics Between Functional-Domains? Proof of Concept". Paper presented at the *CMHR Sandwich Seminar*. Centre for Mental Health Research, Australian National University.

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Kiely, K. M. (September 2009). "Dataset Version 2: Management, Updates and Process". Paper presented at the *Inaugural DYNOPTA Retreat.*

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Kiely, K. M. (2008). "Combining 9 Longitudinal Studies of Ageing. An Overview of Harmonisation Methodologies". Paper presented at the *WIP Day Presentation*. National Centre for Economic and Population Health, The Australian National University

Kiely, K. M. (2008). "The DYNOPTA Dataset. Format and Data Management". Presentation to the *DYNOPTA Scientific Committee Meeting*. Centre for Mental Health Research, The Australian National University

Kiely, K. M. (2008). "Introduction to DYNOPTA". Presentation to the *Joint DYNOPTA Scientific and Steering Committee Meetings.* Centre for Mental Health Research, The Australian National University

Kiely, K. M. (2007). "DYNOPTA Cohort Profile". Presentation to the *DYNOPTA Joint Scientific and Steering Committee Meetings*. John Curtin School for Medical Research, The Australian National University

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CHAPTER 1: Epidemiology of Age Related Hearing Loss and its link with Cognitive Decline

Synopsis

This opening chapter lays the rational for the key research questions guiding this thesis, which aims to document the extent to which sensory and cognitive impairment affects the older population, and inform a better understanding of how these two systems interact. The key motivation for this research is the recognition that hearing and cognition are important for maintaining daily functioning in older adults and their decline with age can have considerable impacts at an individual and societal level. The chapter briefly reviews the literature from epidemiological and psychological perspectives. The epidemiological perspectives presented are chiefly concerned with population burden and risk factors for hearing loss and its comorbidity with cognitive impairment. While the psychological perspectives presented focuses on normal ranges of functioning, and the attempt to identify mechanisms that explain hearing-related cognitive decline.

1.1 Introduction

Global reductions in fertility and mortality rates have resulted in a historically unprecedented demographic transition towards an aged population (United Nations, 2010). Within Australia, increases in life expectancy have outstripped the OECD average, with current estimates placing Australian life expectancies at 79.5 years for men and 84 years for women (Australian Bureau of Statistics, 2012b). In 2006, 13% of the Australian population were aged over 65; this is projected to almost double to between 23% and 25% over the next 50 years. Furthermore, the proportion of adults aged 85 years and older is expected to increase at an even faster rate, growing from 1.6% to between 4.9% and 7.3% by 2056 (Australian Bureau of Statistics, 2008). From an individual perspective, greater longevity means we are faced with new experiences and hold expectations that were rarely encountered by previous generations. A top-heavy population age structure also presents a unique public health challenge to policy makers, as they endeavour to optimise health and well-being among older adults and promote successful ageing (Louria, 2005; Prime Minister's Science Engineering and Innovation Council, 2003)[1].

Sensory and cognitive functions are important abilities that underpin ageing well as they enable older adults to remain engaged with the world around them. Unfortunately, as a consequence of both normative morphological changes and disease processes, deterioration in sensory discrimination is near ubiquitous (Rybash , Roodin, & Santrock, 1991) and many cognitive factors also decline with age (Salthouse, 2012).

[1] While I recognise that there is some contention concerning the most appropriate way to conceptualise successful ageing, it is beyond the scope of this thesis to review this literature. Nevertheless, it is important to acknowledge that a definition of successful or optimal ageing should entail more than mere absence of disease and dysfunction, but also consider a person's opportunities for growth, capacities to adapt and maintain a sense of purpose in life.
(Rowe & Kahn, 1997)
(Baltes & Baltes, 1990)
(Baltes & Carstensen, 1996)
(Bowling & Dieppe, 2005)

Aside from the obvious consequences this has for daily functioning and the limitations placed on the capacity to adjust to late-life transitions (Heyl & Wahl, 2011; Wallhagen, Strawbridge, Shema, Kurata, & Kaplan, 2001), there is also good evidence to suggest that sensory and cognitive ageing occurs in unison. This latter observation has sparked considerable discussion on how best to explain this association (Anstey, Hofer, & Luszcz, 2003a; Baltes & Lindenberger, 1997; Birren, Botwinick, Weiss, & Morrison, 1963; Li & Lindenberger, 2002; Schneider & Pichora-Fuller, 2000). Accordingly, research groups from across a number of disciplines have examined exactly how these two broad functional domains are inter-related across the latter stages of the life-span. A broad range of sensory domains have been incorporated into this research program, though arguably an emphasis has been placed on visual functioning, and perhaps to a slightly lesser extent on hearing.

The opening quotation on the inside cover of this thesis (Beethoven 1802, cited in Comini, 2008) alludes to a central tenet of this thesis, that hearing is of vital importance for social engagement, and its loss can have subtle yet serious impacts on a person's ability and willingness to actively participate socially. Furthermore, there has been little acknowledgment of the serious health issues that poor hearing presents to older adults (Lin, 2012). Perhaps to in some way redress this, there has been a notable resurgence of discussion on how to conceptualise the connection between age-related hearing loss and cognitive functioning within medical, psychological and epidemiological disciplines (Beck & Clark, 2009; Lin, 2011a). Some of this discussion has fallen under the inter-disciplinary banner of Cognitive Hearing Science (Arlinger, Lunner, Lyxell, & Kathleen Pichora-Fuller, 2009), which has recently begun a biennial conference in Linköping, Sweden (Linnaeus Centre HEAD, 2011).

For these reasons, it is with a focus on hearing abilities that this thesis seeks to extend our understanding of age-related sensory decline, by investigating links between peripheral hearing and cognition among older adults from both epidemiological and psychological perspectives. The purpose of this opening review chapter is to provide a rationale for the main research objectives of this thesis. I will take an inter-disciplinary life course perspective that draws upon clinical, epidemiological, biological and psychological literature to briefly describe the nature of age-related hearing loss at both a societal and individual level. I will then discuss how sensory and cognitive systems interact in old age and explain how these patterns of change fit within key theoretical frameworks, before outlining the specific aims of my research.

1.1.1 Population Burden

Sensory and cognitive impairment disproportionately affect older adults and are the leading contributors to the total global burden of chronic disease in people aged over 60. They are the two leading causes of Years Lived with Disability (YLD) in older Australians (Figure 1.1; See also Begg et al., 2007), while hearing loss itself is widely considered the most prevalent chronic condition among older adults (Kiely, Gopinath, Mitchell, Browning, & Anstey, 2012), occurring in at least 80% of American adults aged over 80 (Cruickshanks et al., 2003; Lin, Thorpe, Gordon-Salant, & Ferrucci, 2011) and estimated to affect 75% of Australians aged over 70 years (Access Economics, 2006). With an ageing population, the overall disability burden of these conditions is expected to grow and there is already evidence of such a trend. The Australian Institute of Health and Welfare (AIHW) (2012a) has reported that of all the major disease group classifications, 'Nervous system and sense disorders' showed the biggest increase in Disability Adjusted Life Years (DALYs) in Australia from 2003 to 2010 .

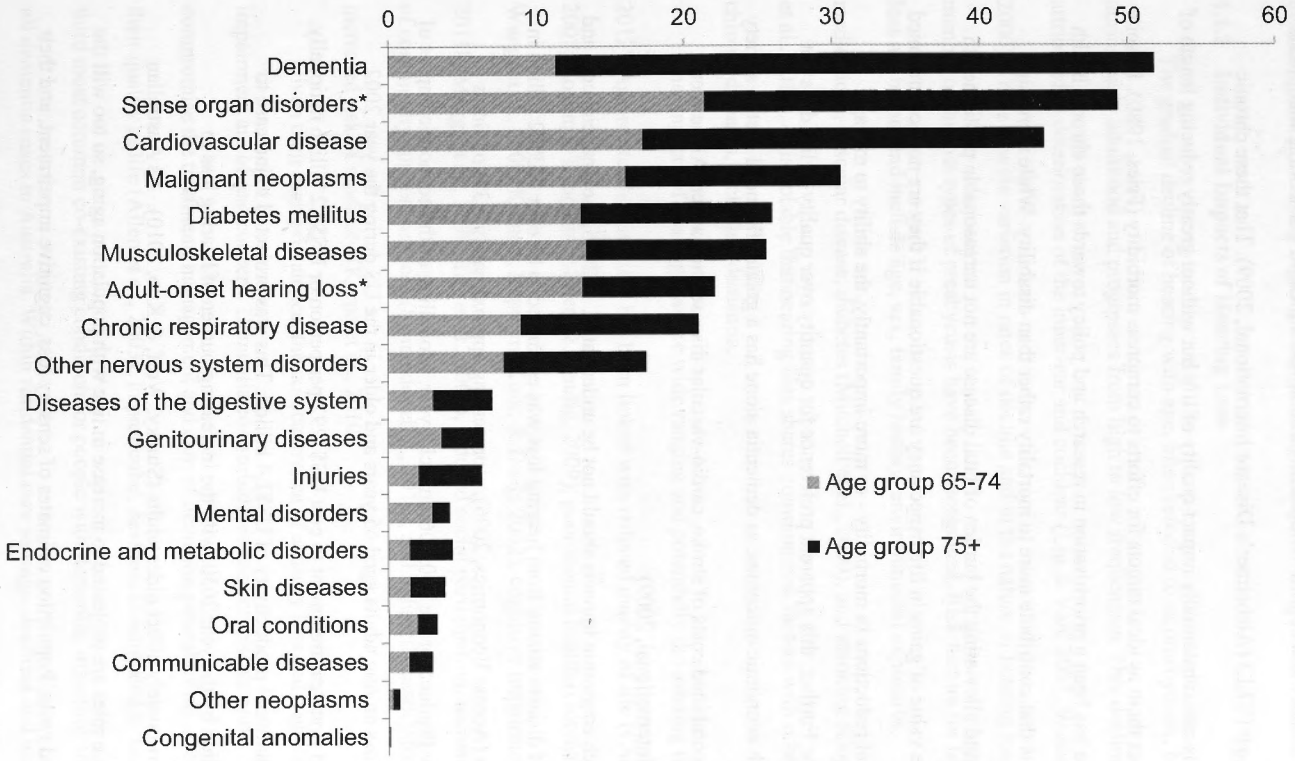


Figure 1.1 Disability Burden (Years Lived with Disability per 1000-person years) in Australian adults aged 65 years and older (Australian Institute of Health and Welfare 2003, author's calculations). *Adult onset hearing loss is a subset of sense organ disorders.

Despite being the predominate contributors to the non-fatal component of DALYs, sensory and cognitive impairment comprise a meagre 1.1% of the total Years of Life Lost (YLL) (Alzheimer's Disease International, 2009). That these chronic conditions can substantially impact quality of life but without greatly reducing length of life, places them as ideal targets for efforts to compress morbidity (Fries, 1980). Despite this, there has been a prioritisation in research and policy towards those chronic health conditions that contribute more to mortality rather than disability. While increasing survival and alleviating the burden of fatal disease are not unreasonable public health goals, the value of gains in life expectancy are questionable if they are not accompanied by parallel reductions in morbidity - or more importantly, the ability to manage morbidity. Further, this apparent preference for quantity over quality of life does not align with economic outcomes, as dementia alone has a greater financial cost to society than the combined costs of stroke, cardio-vascular disease and cancer (Alzheimer's Disease International, 2009).

Such economic impacts should not be understated. In 2005, the financial cost and burden of disease arising from hearing loss was estimated to be over AU$22 billion in Australia (Access Economics, 2006), representing approximately 2.3% of Gross Domestic Product for the 2005-06 financial year. Modelling of the economic impact of hearing loss among adults aged 65 years and older in the US during the year 2002 estimated first year treatment to cost US$1292 per person or US$8.2 billion nationally, and lost national productivity of US$1.4 billion. This was projected to increase to US$9billion by the year 2030, with the increasing burden of hearing loss to disproportionately affect older adults (Stucky, Wolf, & Kuo, 2010). As Australian prevalence rates are projected to increase in line with population aging, so too will the associated costs. Population estimates of sensory loss, cognitive impairment, and their

co-morbidity should therefore be of interest to public health as they are important for informing and prioritising health policy planning decisions.

1.1.2 Individual Impacts of Hearing Loss

The gradual decline of hearing with age, often referred to as presbycusis, begins during early adulthood and progresses from high to low frequencies. This decline is attributed to deterioration of the inner-ear and cochlear (Liu & Yan, 2007; Weinstein, 2000). There is wide variation in rates of decline and in the nature of hearing loss, and a number of distinct types of presbycusis have been recognised. Risk factors for hearing loss are broad, and include age, sex, family history, environmental exposures, medications, vascular disease, diabetes (Mitchell et al., 2009), and smoking (Gopinath et al., 2010). It is notable that hearing loss shares common risk factors with other chronic diseases, including dementia.

The impacts of hearing loss are wide ranging and potentially devastating (Lin, 2012). Age-related hearing loss has been linked with reduced quality of life (Chia et al., 2007; Hogan, O'Loughlin, Miller, & Kendig, 2009), poor mental health (Gopinath, Wang, et al., 2009; Kramer, Kapteyn, Kuik, & Deeg, 2002), cognitive impairment (Lin, 2011b; Maggi et al., 1998; Tay et al., 2006), reduced social participation, increased use of community support services (Schneider et al., 2010), and indirectly associated with increased risk of mortality (Karpa et al., 2010).

Given their high prevalence, the co-occurrence of sensory loss and cognitive impairment are of real concern. Dementia co-morbidities can complicate and compromise the treatment, management and care of dementia patients, thus affecting their quality of life (Allen et al., 2003). In particular, deafness is estimated to be the third most common co-existing condition for people with dementia, affecting 36.8% of all dementia cases in Australia. Within residential care settings, deafness and hearing loss are actually the most common co-existing conditions with dementia (Australian

Institute of Health and Welfare, 2012b). It appears that hearing loss may even be a precurser of dementia (Gates, Anderson, McCurry, Feeney, & Larson, 2011; Lin, Metter, et al., 2011), so their co-occurrence should not simply be considered a coincidence of ageing. The findings of Lin and colleagues (2011b) are particularly startling as they demonstrate that peripheral hearing abilities are independent predictors of dementia incidence. Whether this association can be attributed to a biological or a social mechanism is unclear, though APOE e4/e4 has recently been linked with higher audiometric hearing thresholds (Kurniawan et al., 2012). Of course this same genotype is one of the strongest single genetic risk factors for Alzheimer's Disease (Corder et al., 1993).

Unfortunately the gradual and progressive nature of hearing loss means it is often unnoticed. Despite its strong impact on health and wellbeing, age-related hearing loss is believed to be both under-recognized and under-treated (Reuben, Walsh, Moore, Damesyn, & Greendale, 1998). It is possible that this is partly due to the misperception that hearing loss is a normal part of the ageing process (Lin, 2012), a view that is reinforced by the near universal experience of hearing difficulties among the oldest old. One way to gain a better appreciation of the extent to which hearing loss can affect individuals, is to compare its impacts to other chronic conditions commonly found among older adults. For example, in the algorithm used to calculate DALYs, moderate hearing loss is considered to have a disability weight comparable to chronic pain (Davis, Smith, Ferguson, Stephens, & Gianopoulos, 2007), while the disability weight for severe hearing loss is comparable to major depressive disorder or pneumonia (Access Economics, 2006). The poor acknowledgment of the real problems hearing loss can cause is reflected in the research literature. Although there are a number of large epidemiological surveys that include gold-standard measures of sensory function (Cruickshanks et al., 2003; Lin, Thorpe, et al., 2011; Luszcz et al., 2007; Mitchell et al.,

2009), recent reviews have found that globally only a small number of surveys were suitable for estimating hearing impairment in the general population (Pascolini & Smith, 2009). There are currently sparse national data on hearing impairment in older people, in Australia or elsewhere, with recent investigations of risk factors for incidence of age-related hearing loss being underpowered (Gopinath et al., 2010; Gopinath, Schneider, Rochtchina, Leeder, & Mitchell, 2009; Mitchell et al., 2009). Existing Australian prevalence of sensory loss has been estimated for broad age cohorts (Access Economics, 2006). In this is the implicit assumption that an adult in their early 70s share comparable needs, expectations and experiences to adults aged in their 90s – an assumption that is unlikely to bear up against scrutiny. It is clear that more precise age-prevalence estimates of hearing loss and its comorbidities are needed.

The practicalities of data collection could be one reason why reliable estimates of age-related hearing loss have been difficult to obtain. Costs and logistics involved in conducting audiometric assessment make clinical measures prohibitive for many epidemiological surveys, so self-report measures or interviewer judgements are often used instead. These self-reported measures of hearing loss have previously been thought to be reliable and predictive of audiometric hearing loss whilst also providing an ecologically valid measure of perceived hearing difficulties (Caban, Lee, Gomez-Marin, Lam, & Zheng, 2005; Nondahl et al., 1998; Sindhusake et al., 2001). Yet there is good evidence to suggest that this is not the case (Hong, Ronis, & Antonakos, 2011). In particular, dual sensory loss estimates are commonly reliant on self-report data, or a combination of visual acuity and a measure of subjective hearing (Smith, Bennett, & Wilson, 2008). This is problematic as self-report data can be biased by individual differences. A recent review on dual sensory loss identified 37 research articles on the prevalence or impacts of dual sensory loss (Schneider et al., 2011). Unfortunately, Schneider and colleagues reported that a lack of consistent standardized definitions and

differences in study age and sex distributions resulted in large discrepancies in reported estimates. They called for more studies on dual sensory loss that use objective clinical measures and longitudinal data. The present thesis seeks to address this shortcoming in the literature.

1.1.3 Psychological Perspectives

By emphasising dementia, pathology, impairment and loss, most of the epidemiological and clinical literature focuses on secondary ageing processes (Anstey, Stankov, & Lord, 1993). But it is also important to understand the inter-relations between hearing and cognition as primary ageing processes. Following these lines, there has been a long tradition within psychology that has investigated links between hearing and cognition along the spectrum of normal functioning. Early researchers attempted to elucidate the extent to which auditory discrimination could be identified with mental abilities (Deary, 1994; Galton, 1883; Raz, Willerman, & Yama, 1987). More recently the focus has shifted to identifying explanatory mechanisms that underlie the age-related interconnection between auditory acuity and cognitive function. This has been driven by a long line of studies demonstrating a strong connection between hearing and cognition among older adults (Anstey, Luszcz, & Sanchez, 2001a; Birren et al., 1963; Lin, 2011a; Lindenberger & Baltes, 1994; Schaie, Baltes, & Strother, 1964). Though not all studies have supported this finding (Colsher & Wallace, 1990), and it is further argued that the unique association may not hold longitudinally (Anstey et al., 2003a; Lindenberger & Ghisletta, 2009).

Explanations of hearing-cognition associations can be distinguished by the direction of the proposed causal relation (Baltes & Lindenberger, 1997; Gallacher, 2004; Schneider & Pichora-Fuller, 2000). Common cause theories attribute the association to a third variable, generally purported to be an unidentified biological mechanism. Though common cause accounts attracted considerable debate during the

late 1990s and early 2000s, more recently such explanations have fallen from favour. Cascade models place sensory loss as the driver of subsequent cognitive decline via a number of pathways (Figure 1.2), including social isolation and increased cognitive load. Social withdrawal due to hearing loss could result in lower participation in stimulating activities purported to help maintain cognitive reserve (sensory deprivation). Similarly, effortful listening may require more cognitive resources to be allocated to lower level sensory processes in order to attend to degraded speech sounds, thus diverting cognitive resources away from higher order processes and compromising cognitive function (sensory degradation). On the other hand, an alternative argument might construe effortful listening as cognitively stimulating for some people, in which case poor hearing may actually predict better cognitive function. Individuals with the capacity to maintain both their hearing and cognitive abilities despite degraded sensory inputs may benefit from the extra effort. Cognitive underload hypotheses make the opposite prediction to cascade models, and suggest that low cognitive function influences sensitivity to sensory information. These hypotheses specify modular models that treat sensation and cognition as distinct constructs that can be distinguished by their contrasting predictions. However, these explanations can be assimilated by a single model that views hearing and cognition as components of a single integrated system (Schneider et al, 2000). Another theoretical framework that can be used to inform our understanding of hearing-related cognitive decline maintains that a more elucidating index of old age is provided by functional ability rather than chronological age. Such biomarker accounts place hearing as a functional biomarker of cognitive function and make similar predictions to cascade models (Anstey, 2008b). Proponents of biomarker theories argue that age is in essence an empty variable (Bytheway, 2005) that serves as a proxy for developmental processes and senescence. This view sits comfortably within a broad research program that has sought to describe late-life developmental phenomena

in terms of processes other than ageing, such as mortality (Gerstorf, Ram, Hoppmann, Willis, & Schaie, 2011), disability, and functional decline (Sliwinski & Mogle, 2008). These theories and their supporting evidence are reviewed in more detail in Chapters 8 and 9, which report on the testing of dynamic longitudinal interrelations between hearing and cognition.

Hearing assistive technologies, including aids and hearing loop induction systems, have improved considerably over the past decade and show great efficacy in counteracting the adverse impacts of hearing loss, especially when incorporated into a holistic rehabilitation program (Lin, 2012). Despite their many benefits, levels of hearing aid use have remained surprisingly low (Gopinath et al., 2011; Sanchez, Scott, Esterman, & Luszcz, 2011). This reluctance to use hearing aids could be due to personality characteristics, costs, awareness of hearing problems, and difficulties in hearing aid acclimatization. Aside from the more obvious benefit of improving communication and quality of life, an intriguing hypothesis germane to this thesis, is that hearing aid use may actually be protective against hearing-related cognitive decline (Lin, 2012). The rationale for this theory is that by facilitating easier communication and removing barriers to social engagement, hearing aids may interrupt the upward cascade of hearing loss to poor cognition (Figure 1.2). An additional novel view could be that the process of becoming accustomed to using a hearing aid could even constitute a type of cognitive training in and of itself. In light of this, one of the final aims of this thesis will be to examine this notion that hearing aids may break the nexus between poor hearing and cognitive decline.

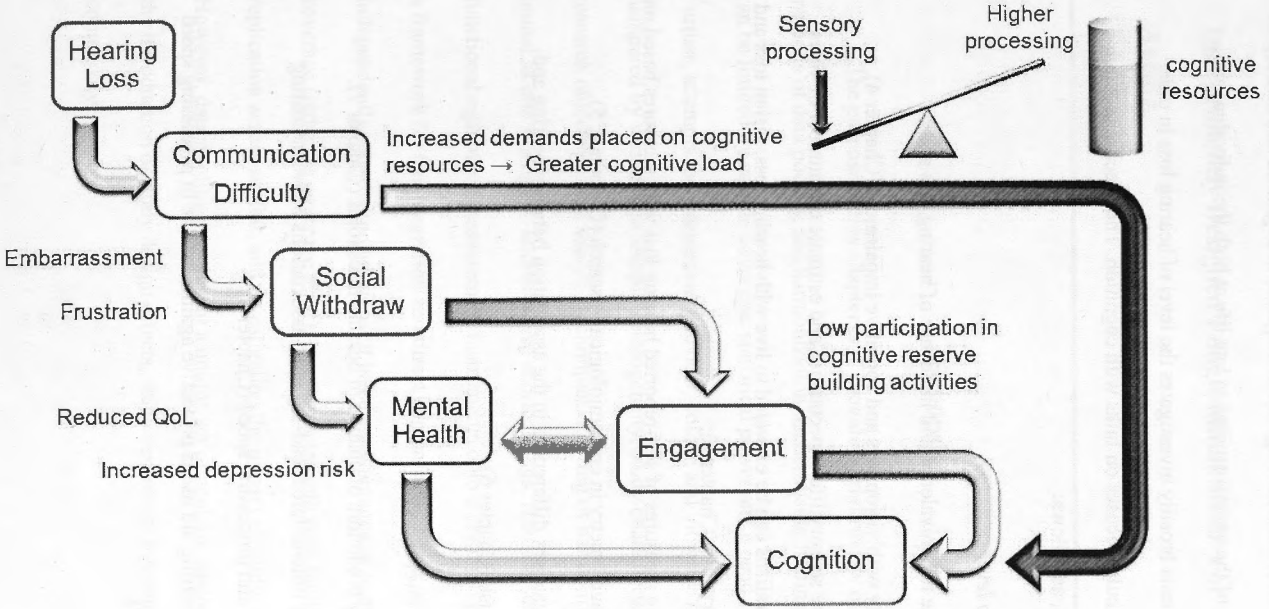


Figure 1.2 A cascade model depicting two bottom-up pathways between hearing loss and cognitive decline, one via a social mechanism (sensory deprivation) and the other via a resource allocation mechanism (sensory degradation).

1.1.4 Primary Objectives and Research Questions

It is important to understand how age-related decline in cognition and sensory function interact as part of the ageing process in late life at both the individual and population level. This thesis broadly investigates the level of hearing loss in older Australians, and in particular focuses on links with cognition. The specific research aims follow two themes, as follows:

1.1.4.1 Population level

- To estimate the prevalence and incidence of hearing loss and its comorbidity with vision loss and cognitive impairment (Chapter 4).
- To calculate sensory life expectancies and estimate the number of years older Australians can be expected to live with hearing loss, vision loss and dual sensory loss (Chapter 5).
- To compare measures of self-reported hearing loss with measures based on pure-tone audiometry in epidemiological research (Chapter 4, 5).
- To investigate sex differences in the association between hearing and mortality risk (Chapter 6).

1.1.4.2 Individual level

- To identify predictors of change in hearing thresholds (Chapter 7).
- To explore time-ordered lead-lag inter-associations between hearing thresholds and processing speed (Chapter 8).
- To test if hearing aid use is protective against decline in processing speed (Chapter 9).

CHAPTER 2: Combining Data from Independently Designed Longitudinal Studies of Ageing

Synopsis

The purpose of this chapter is to provide a brief review of the rationale and methods of data pooling and harmonization. Generalising findings from single studies can be limited by sample coverage, statistical power and a need for replication. Further, attempts to systematically review, collate and integrate research findings can be hampered by variation in sample heterogeneity, study characteristics and measures, research objectives, and analytic techniques. Pooling of harmonized data has been touted as one strategy to address these shortcomings. Harmonisation is the rescaling of functional equivalent measurement instruments onto a common metric, which provides a framework for the integration and direct comparison of data obtained from independently sampled populations. It has the advantages of increasing population coverage (reducing coverage error), increasing sample size, facilitating instantaneous replication across studies, and investigating the impact of study idiosyncrasies. However, data pooling can present a unique set of analytic challenges. These include the introduction of study design effects, study censoring, information loss, and dataset complexity.

2.1 Introduction

An initial key focus of this thesis was to examine the utility of harmonised and pooled data for testing theories of cognitive ageing. However, as my research evolved the aims shifted to a more substantive focus with less emphasis on the methodological issues that may arise when establishing a common ground for combining population based longitudinal studies of ageing. Nevertheless, this thesis does draw upon pooled data from two studies to inform some of the research questions posed in the opening chapter. It is therefore important that such methodological issues are acknowledged and addressed. The purpose of this chapter is to make the case for data pooling, outlining the benefits and different approaches to combining studies. It will also highlight some of the challenges of analysing pooled data and describe potential solutions to address these issues.

2.1.1 The Case for Pooling Data

2.1.1.1 Enabling synthesis

Research synthesis is fundamental for accumulating evidence based knowledge, and key to the formation and progression of scientific consensus (Curran, 2009; Light & Smith, 1971). This is typically reached firstly via replication, and then by conducting systematic reviews and meta-analyses of published (and if available, unpublished) estimates. However, there is a view that opportunities for assimilating psychological and gerontological research have been limited because of disparate research agendas and incompatible study characteristics (Piccinin & Hofer, 2008). To support their claim, Piccinin and Hofer cite a review of the literature on age-related cognitive decline that was restricted to a qualitative narrative (Park, O'Connell, & Thomson, 2003). Park's intended meta-analysis could not be conducted because of heterogeneous samples, variability in assessment procedures and differences in time intervals between data

collection periods across the selected studies. Comparability problems can also arise due to a lack of commonly applied standards or definitions. The lack of a uniform definition for functional decline prevented a more precise integration of study findings that would otherwise have facilitated a closer inspection of causal pathways leading to frailty and disability in late life (Stuck et al., 1999). Similar issues concerning non-standardised definitions have arisen when reviewing prevalence estimates of dual sensory loss (Smith et al., 2008).

2.1.1.2 Limits of single sample designs

An undue emphasis on significance testing, over reliance on single-sample studies and small sample sizes (Dyer, 1986; Schmidt, 1996) can constrain generalizability of research findings and slow the accumulation of scientific evidence (Curran, 2008). Within psychology in particular, there is currently considerable debate concerning replication, with a special issue devoted to the topic in a recent edition of Perspectives on Psychological Science (Pashler & Harris, 2012; Pashler & Wagenmakers, 2012) and articles in both Science (Carpinter, 2012) and Nature (Yong, 2012). These authors have expressed a belief that some sectors of the discipline have stagnated and face credibility issues due to a failure to reproduce, or even attempt to reproduce, a large number of published findings. This has called into question the veracity of un-replicated research and its theoretical underpinnings (Ferguson & Heene, 2012). Such reflection has underscored a strong push to recognise the importance and afford greater credence to research replication (Frank & Saxe, 2012). Normative data are often derived from small samples that have been recruited via non-random methods, which have the potential to bias estimates. Even in cases where normative data has been drawn from random sample designs, the sample frame may be limited in its population coverage (Kiely et al., 2011). Cross population comparisons are important for investigating universal ageing processes as well as identifying regional differences. Such comparisons can be

made both within and between national and cultural populations. Of note, there are a number of longitudinal studies of older adults within Australia, and although all of these are used to draw inferences about the broader Australian population, few actually sample the entire national population (e.g. Dunstan et al., 2002; Wooden, Freidin, & Watson, 2002). Most individual studies sample smaller regions (Anstey, Christensen, et al., 2011; Banks et al., 2008), cities (Luszcz et al., 2007) or suburbs (Mitchell et al., 2009). The requirement for longitudinal evidence is also a factor explaining the slow progress in disciplines orientated by a lifespan perspective (Piccinin & Hofer, 2008). As longitudinal studies are expensive to run, researchers have an obligation to funding bodies and to study participants to maximise investment returns (Anstey, Bielak, et al., 2011b). Thus, an over-emphasis on single study designs has broad implications for a number of common research aims, including the estimation of normative data, drawing cross-population comparisons and discerning chance findings from real phenomena.

2.1.1.3 Pooling as a solution

Researchers from a range of disciplines with an interest in population ageing and gerontology have advocated data pooling as a good strategy to address these shortcomings (Aijanseppa et al., 2005; Anstey, Byles, Luszcz, Mitchell, Steel, Booth, Browning, Butterworth, Cumming, Healy, Windsor, Ross, Bartsch, et al., 2010; Bath, Deeg, & Poppelaars, 2011; Cooper & Patall, 2009; Dyer, 1986; Hofer & Piccinin, 2009; Kiely et al., 2011; Minicuci et al., 2003; Ofstedal et al., 2007; Piccinin & Hofer, 2008; Schenker & Raghunathan, 2007; Yunhwan & Shoji, 2003). Firstly, combining studies allows researchers to make more precise descriptions of the ageing population, and draw stronger inferences concerning determinates and impacts of ageing processes. It will enhance population coverage, allowing for cross-population comparisons, and enables the estimation of more generalizable population norms. Pooling also increases statistical power, which may allow researchers to conduct more reliable investigation of

under-represented groups, as well as take full advantage of recent methodological advancements in longitudinal modelling. Further, by aggregating data from existing studies researchers can not only improve comparability across studies, but also establishes a framework that better aligns and coordinates research agendas. This has the advantage of providing instantaneous replication and therefore quickly builds the evidence base required to inform scientific consensus. Pooling projects will typically bring together researchers from across disciplines, institutions, and countries, thus fostering collaboration and knowledge sharing between research teams who may otherwise work in isolation. This has the added advantage of providing an inventory of existing longitudinal studies, which will help highlight existing research gaps (Anstey, Kiely, et al., 2011).

2.1.2 Variable Harmonization

For data pooling to be effective it is crucial that aggregated studies have compatible data. The easiest way to achieve this is to coordinate study designs in the development stage so they collect the same measures and follow the same sampling procedures. However, in the case of independently designed studies, it is unlikely raw data can be directly pooled so variables must be retrospectively reconstructed via process often referred to as harmonization. Variable harmonisation is a technique of response conversion that is predicated on the criterion of equivalence (van Buuren, Eyres, Tennant, & Hopman-Rock, 2001), the degree to which different measurement instruments index identical phenomena (van de Vijver & Tanzer, 2004). Importantly, commensurate measurement indices that are considered equivalent should not exhibit bias towards a particular population.

2.1.2.1 Measurement equivalence

Measurement equivalence refers to equality of measures and requires that two measures are scored on the same response scale. Psychometricians provide a mathematically rigorous definition of measurement equivalence in terms of probabilities. Specifically, individuals with equivalent latent ability have the same distribution of response probabilities (Meredith, 1993) (See also Bontempo & Mackinnon, 2006). This stipulates that two individuals with the same ability levels should provide the same response to the same items. In the context of psychological or cognitive assessment, this occurs when identical instruments are employed by each survey. For example, if two surveys administer the Mini Mental State Examination (MMSE) (Folstein, Folstein, & McHugh, 1975a), this will meet the criteria for measurement equivalence. In principle such survey data should be directly comparable and harmonisation would not be necessary prior to pooling of raw data.

2.1.2.2 Functional equivalence

Although measurement equivalence is the ideal for data pooling, it is more likely that pooled studies share similar but non-identical measurement instruments. In this scenario harmonisation can be justified when functional equivalence is demonstrated. The criterion of functional equivalence is met when distinct measurement instruments assess the same construct dimensions. For this reason, functional equivalence is considered the primary prerequisite for variable harmonisation (Hoffmeyer-Zlotnik & Wolf, 2003) and should enable cross-population comparisons.

Evaluation of the functional equivalence of different instruments requires consideration of item compatibility and the format of the response scale. It must be shown that items are worded in a way that will be interpreted by participants as being conceptually the same. For example, self-rated hearing may be assessed by asking a respondent a questions concerning general hearing 'difficulties', 'problems' and 'losses', or questions concerning hearing limitations in specific situations, such as

hearing over background noise, or following a conversation in a busy restaurant. For these items to be pooled it must be demonstrated that such items are interpreted in the same way by respondents. Even when items clearly refer to the same construct, such as 'self-rated health', changing the frame of reference from 'in general' to 'for your own age' or 'compared to when you were younger', will alter response patterns (Sargent-Cox, Anstey, & Luszcz, 2008). Likewise, it unlikely to be reasonable to compare an item with a short frame of temporal reference 'in the past two weeks' with an item with a longer frame of temporal reference "in the past two years" as the former is likely to be more sensitive to transient state based process whereas the latter will be sensitive to more permanent trait-like processes. For cross-national research an additional layer of complexity is added by the need to ensure interpretation of survey question is equivalent across languages (Bath et al., 2011). Once functional equivalence has been established a conversion key for variable rescaling must be created (van Buuren et al., 2001; van Buuren, Eyres, Tennant, & Hopman-Rock, 2005), and there are a number harmonisation methods available for achieving this.

2.1.3 Methods of Harmonisation

There are two main methods for creating a conversion key for retrospective variable harmonisation. The simplest approach is to recode or collapse response categories, this is sometime referred to as 'by fiat' harmonization. A more mathematically rigorous technique is to estimate a latent construct with a model based approach. Deciding upon the appropriate method for harmonisation will depend on available data and variable response scales.

2.1.3.1 By fiat

Harmonisation by fiat is the recoding of responses from unique items onto a common metric. This requires decisions on variable reconstructions are made by a panel

of informed experts (van Rijckevorsel, van Buuren, & de Kleijn – de Vrankrijker, 2001). This approach has been used in a number of cross-national studies as part of the Europe Comparison of Longitudinal European Studies on Ageing (CLESA) project which has reported on pooling of self-rated health (Bardage et al., 2005), activities of daily living (Nikula et al., 2003; Pluijm, Bardage, Nikula, Blumstein, Jylhä, et al., 2005) and depressive symptoms (Bath et al., 2011; Pluijm, Bardage, Nikula, Blumstein, Jylhä, et al., 2005). .

Scale type is an important factor when considering the reliability of harmonisation by recoding response categories. Continuous measures will meet the criteria of equality of measures and so would normally be directly comparable posing few problems for harmonisation or pooling. Conversion may be needed if original measurements exist on different scales, for example converting imperial units to metric units. Other adjustments may be necessary to account for study differences in devices and equipment used to make biometric measurements. Recoding of nominal scales will generally be robust, if a common set of mutually exclusive categories can be defined. Examples of such variables include marital status, career occupation and highest qualification attained. In contrast, optimal rescaling of ordinal and interval measures can be ambiguous, particularly if sampled populations are not matched on key variables (e.g. age, sex, education). Further, it is possible that response propensities can be influenced by the number of response options available. Because of ambiguity in collapsing original response categories onto a common scheme, harmonisation 'by fiat' is recommended only when there is little possibility of dispute, original categories are finely grained and conversion has expert endorsement (Bath et al., 2011; van Buuren et al., 2001).

2.1.3.2 Model based approaches

A superior method of harmonising data by response conversion, especially for variables with ordinal and interval scale properties, is to fit a statistical model which draws upon the principles of Item Response Theory (IRT) or Latent Trait Analysis. IRT is a family of statistical techniques designed for the analysis of item characteristics and latent abilities (Orlando, Sherbourne, & Thissen, 2000). Compared to classical test theory, IRT provides a more sophisticated and powerful psychometric method for relating discreet observations to an underlying latent variable. IRT has wide ranging applications in psychological measurement (Embretson, 1996; Lord & Novick, 1968), however integrative researchers looking for robust modelling of harmonised data will be specifically interested in the capacity for systematic item and instrument linkage (Embretson, 1996; Lim, 1993; Pommerich, Hanson, Harris, & Sconing, 2004). Latent variable approaches have been used to harmonise measures of Activities of Daily Living (van Buuren et al., 2001; van Buuren et al., 2005), memory and verbal abilities (McArdle, Grimm, Hamagami, Bowles, & Meredith, 2009) and depression (Orlando et al., 2000). In one extreme example, Salthouse (2004, 2005) analysed pooled experimental data from over 30 studies to investigate the factor structure of intelligence and executive function in older adults. IRT and latent variable approaches can be preferable to the by fiat method because the determination of response scale cut points are not arbitrary but informed by structural relationships present in observed data. However, as a means to translate equivalent instruments onto a latent scale, IRT is only possible when a linkage or anchor point is provided by a common instrument across studies. Without data overlap latent variable harmonisation is not achievable. The requirements for linkage points and asymptotically large samples may mean that opportunities for harmonisation via latent modelling are limited, and in some instance additional linkage studies may be needed.

2.1.4 Challenges of Data Harmonization and Pooling

2.1.4.1 Study censoring

Study censoring is a mechanism for missing data which is unique to large harmonised and pooled datasets. It arises when a particular study does not collect a variable of interest. Since in this instance the reason for study censoring is known (the item was not collected), this data can be classified as missing at random and is therefore non-ignorable (Little & Rubin, 2002). Normally study censoring would simply mean that variables cannot be harmonised and either the variables or the censored population must be excluded from analyses. However, for key variables which are likely to regularly feature as outcome variables or modelled as contextual covariates, it may be justified to derive estimates of the study censored data from other responses. For example, age left school could be imputed based on a person's age, sex, qualifications and occupation (Anstey, Bielak, et al., 2011b; Kiely, Anstey, & The DYNOPTA Investigators, 2009). Another example is provided by the harmonisation of ADL data in the CLESA project, where toileting was inferred by responses to bathing and sitting/rising from a chair (Pluijm, Bardage, Nikula, Blumstein, Jylha¨m, et al., 2005).

2.1.4.2 Information loss

Variable harmonisation can come at the cost of information loss. Variables harmonised 'by fiat' will always be restricted to the lowest common denominator of variable coding thus reducing variability in the data (Glover, 1996). In other words, the contributing studies raw data may retain finer grained measures allowing for more nuanced and complex analyses but these will not be necessarily directly comparable. So any gains made by increased sample size and enhanced population coverage may be countered by the cost of information loss. In a related vein, increased sample size also

has the potential to overpower analyses so statistically significant results could be found for weak associations.

2.1.4.3 Dataset complexity and study design effects

A further challenge to working with pooled longitudinal studies is dealing with the added complexity of aggregated data. Largely because of missing data arising from non-response, attrition and mortality, multivariate data from single longitudinal studies can be difficult to work with (Chatfield, Brayne, & Matthews, 2004). It is important to make correct decisions regarding the appropriate modelling of time. Longitudinal analyses can also be limited if measures are not consistent within studies across waves. These difficulties will be compounded when a multiple longitudinal studies are harmonised and pooled. When analysing pooled longitudinal data, consideration must be given to irregular time schedules, age and cohort effects, sample composition and measure availability. Another cautionary note concerns increased statistical power.

Study design effects refer to differences in data collection and coding that have the potential to influence findings. This variation presents new sources of error not normally encountered in single sample survey research (Schenker & Raghunathan, 2007). Study effects can arise from variability in sampling procedures, including sample frames (e.g. electoral role, Medicare), sampling units (e.g. household, person), geographic coverage and response rates. In anticipation of low response rates, it is common for longitudinal studies to over sample particular segments of the population, or recruit additional household members who are related to the original sample unit. These factors can result in skewed samples that are not true one to one reflections of the original population. This could be problematic if the purpose of pooling has been to conduct epidemiological research and draw inferences concerning inter-population or intra-population characteristics. Study design effects therefore reflect the inflation of sampling variances relative to simple random sampling. Other sources of study effects

include differences in missing data codes, interviewer protocols and administrative procedures. More subtle study effects could even arise from the ordering in which measures are administered. The concern is that if not properly accounted for, study design effects could either mask true effects or result in spurious associations.

When the aim of harmonization and data pooling is to facilitate cross-population comparisons then study differences will be of substantive interest. However, if the purpose is to enhance coverage of a distinct population, then study differences will become nuisance variables. In either case it is important to minimise any statistical noise that could be introduced by design characteristics so that any study effects reflect true differences between populations. The key point is this: When analysing aggregated data it may not be enough that all variables have been harmonized to be functionally equivalent. Further steps will have to be taken to remove bias introduced by survey design. The harmonisation literature has not yet explored how to best attenuate such design effects, though a number of approaches are open to researchers.

2.1.5 Minimizing Study Design Effects

The simplest approach to address study effects when analysing harmonised and pooled data is to adjust for study as a covariate (e.g. Fauth, Gerstorf, Ram, & Malmberg, 2012; Wang et al., 2003). Adjusting for study is generally more appropriate when the study samples are drawn from the same cultural and national context, and also share common survey protocols and design parameters. This is most likely to occur when the same research team has worked across each of the individual studies. This was the case when Fauth and colleagues (2012) analysed change in depression across four Swedish longitudinal studies. If a large number of studies are pooled, it is also possible to use hierarchical linear modelling where persons are nested within study.

In some instances, rather than adjust for study effects, individual study estimates have been presented alongside combined estimates. This approach has typically been

adopted when calculating prevalence or incidence rates. For example, the Blue Mountain Eye Study and the Melbourne Visual Impairment Project pooled best corrected visual acuity data to provide 30 year population projections of visual impairment in Australia (Foran, Wang, Rochtchina, & Mitchell, 2000). An alternative strategy when calculating prevalence from pooled data is to apply population weights in addition to adjusting for study effects (Anstey, Burns, et al., 2010). Population weights can be used to combine studies by accounting for differences selection probabilities that lead to different segments of the population being under or over represented.

A third option is to conduct patient level meta-analysis (Dyer, 1986). This method has most recently been advocated by Hofer and colleagues as 'integrative data analysis' (Hofer & Piccinin, 2009; Hofer & Piccinin, 2010). Under this approach variables are still harmonised so as to remain comparable across datasets, but rather than analyse the pooled data as a single dataset, the same analytic model is run on each individual study and the resulting estimates are pooled. This has the benefit over normal meta-analysis of published data as identical analytic models are run in parallel on comparable data and so are better co-ordinated or 'integrated'. However, there has been little published research using this approach.

2.1.6 Conclusion

Data pooling allows researchers to make the most of existing data and foster collaboration, streamlining research programs and coordinating research objectives. Although pooling may enhance coverage and increase statistical power, this can come at the cost of diminished discriminatory power (information loss), the introduction of study design effects and study censoring on key variables. For longitudinal studies, this can result in complex unbalanced formats with a high proportion of missing data. It is clear that steps must be taken to minimise these limitations, and this can be accomplished via adjusting for study effects, weighting each study to address sampling

bias or employing patient level meta-analytic techniques. A secondary objective of this thesis is to demonstrate the use of, and evaluate the utility of, harmonised longitudinal cognitive and sensory data.

CHAPTER 3: Study Sample and Measures

Synopsis

The Dynamic Analyses of Optimise Ageing (DYNOPTA) project has harmonised and pooled nine epidemiological studies of human ageing to examine pathways to compressing morbidity and optimise healthy ageing in the Australian population. Two contributing studies, the Australian Longitudinal Study of Ageing (ALSA) and the Blue Mountains Eye Study (BMES), collected clinical measures of hearing, vision and cognitive function between 1992 and 2004. This chapter focuses on describing the participants, sampling methods and data collection procedures of these two studies, and outlines the measures that are used in the subsequent chapters of this thesis. This chapter also highlights some of the issues that must be addressed when working with pooled longitudinal data, making reference to the broader DYNOPTA sample.

3.1 Background

All studies reported in this thesis draw on data from the Dynamic Analyses to Optimise Ageing (DYNOPTA) project (Anstey, Byles, Luszcz, Mitchell, Steel, Booth, Browning, Butterworth, Cumming, Healy, Windsor, Ross, Burns, et al., 2010). DYNOPTA is a cross-institutional and inter-disciplinary project that has harmonised and pooled nine Australian Longitudinal studies of ageing. The broad aims of the project are "to identify effective pathways to compressing morbidity and optimizing ageing" (page 44, Anstey et al., 2010). The pooled dataset is rich including five theme areas of cognitive functioning, sensory-motor functioning, mental health, mobility and functional independence, and mortality. The pooled dataset also includes background variables that cover socio-demographics, health, lifestyle and medical conditions.

This methods chapter will briefly describe the broader DYNOPTA sample, and provide more detailed information on the design and sample characteristics of individual DYNOPTA studies that contributed sensory and cognitive measures to the pooled dataset. The chapter will explain how the variables presented in this thesis were harmonised. It will also provide basic cross sectional and longitudinal descriptive data for the key outcome variables and identify the appropriate scaling of time for longitudinal analyses that will be presented in subsequent chapters. An ancillary aim of this chapter is to demonstrate some of the considerations that must be made when pooling and analysing harmonised data.

3.1.1 The DYNOPTA Dataset

DYNOPTA provides the contextual backdrop of the research presented in this thesis. The full DYNOPTA dataset is large and complex, consisting of 50,652 participants followed longitudinally on up to 11 measurement occasions over a 15 year period. With over 400 variables there are in excess of 18 million data points. The target

population for DYNOPTA was defined as all Australians born prior to December 1955. However, target populations for individual studies varied. The Household, Income, Labour Dynamics of Australia (HILDA) (Wooden et al., 2002) and the Australian Diabetes and Obesity and Lifestyle Study (AusDiab) (Dunstan et al., 2002) both sampled the national Australian population of age 15 years and older. The Australian Longitudinal Study of Women's Health (ALSWH) (Lee, Dobson, et al., 2005) was also a national study, however it sampled only women in two narrow age bands. In contrast the Australian Longitudinal Study of Ageing (ALSA) (Luszcz et al., 2007), The Blue Mountains Eye Study (BMES) (Tay et al., 2006), Canberra Longitudinal Study (CLS) (Christensen et al., 2004), Melbourne Longitudinal Studies of Ageing (MeLSHA) (Browning & Kendig, 2010), Personality and Total Health (PATH) Through Life Survey (Anstey, Christensen, et al., 2011) and the Sydney Older persons Study (Broe, 2003) sampled smaller metropolitan areas.

The DYNOPTA study design illustrates some of the issues raised in the previous chapter concerning the complexities of analysing pooled longitudinal data. Figure 3.1 shows the interview schedule for all contributing DYNOPTA studies and Table 3.1 shows the studies which collected measures that could be drawn upon to inform the substantive research questions posed by this thesis. There are notable study differences in time intervals between waves and the years of study commencement. There are differences in age-range, for example PATH study has a narrow 5-year birth cohort, whereas the BMES has a broad age cohort (ages 45 and older). Due to overlapping sample frames it is possible for some participants to have participated in multiple studies. For example, a 90 year old adult recruited to the HILDA study at in the year 2000, could also have participated in the baseline wave of the CLS 12 years prior at the age of 78. Analyses of these pooled data must therefore consider the appropriate

Figure 3.1 Interview schedule for all contributing DYNOPTA studies. Studies included in this thesis are ALSA, and BMES.

Table 3.1 Data availability for potential outcome variables considered for this thesis, by contributing study and wave.

Study	Wave	Age range	Vision		Hearing		Cognition					
			SR	VA	SR	PTA	MMSE	NART	BNT	Speed	WAISsim	Fluency
ALSA	1	65-103	X	x	x	x	x	x	x	x	x	.
	3	66-105	X	x	x	x	x	x	x	x	x	x
	6	72-101	.	x	x	x	x	x	x	x	x	x
	7	75-102	.	x	x	x	x	x	x	x	x	x
SOPS	1	75-97	.	x	x	.	x	x*	x	.	x	x
	2	78-99	.	x	.	.	x	x	x	.	x	x
	4	80-101	.	x	.	.	x	.	x	.	x	x
	5	84-106	.	x	.	.	x	.	x	.	x	x
CLS	1	70-103	X	.	x	.	x	x		x	x	x
	2	74-102	X	.	x	.	x	x		x	x	x
	3	78-101	X	x	x	.	x	x		x	x	x
	4	82-105	X	x	x	.	x	x		x	x	x
BMES	1	45-100	X	x	x	.	.	.	.	.	.	.
	2	50-98	X	x	x	x	x	.	.	.	.	.
	3	55-99	X	x	x	x	x	.	.	.	.	.
PATH	1	60-66	X	x	.	.	x	.	.	x	x	.
	2	64-70	X	x	x	.	x	.	.	x	x	.

SR: Self report data including ratings of general functioning and impairment on iADLs; VA: Visual Acuity; PTA: Pure Tone Thresholds; MMSE: Mini Mental State Examination; NART: National Adult Reading Test (*Schonell in SOPS wave 1); BNT: Boston Naming Task; Speed: Processing Speed; WAISsim; Wechsler Adult Intelligence Scale Similarities Task; Fluency: FAS and Animal Word Generation Task

definition of time and take steps to minimise cohort effects. Study censoring is also clearly evident for some content areas. For example, while the SOPS includes broad coverage of cognitive measures, it lacks comprehensive data on sensory functioning which limits the scope of the SOPS sample to address any research questions regarding links between age-related cognitive decline and sensory loss. Unsurprisingly, research projects using DYNOPTA data have been restricted to the use of a sub-set of contributing studies. For example, a study investigating the levels of visual and cognitive impairment among older drivers was restricted to using ALSA, BMES and SOPS (Ross et al., 2009).

After initially planning to use all studies presented in Table 3.1, it was decided to narrow the scope of the current thesis to hearing loss, vision loss and cognitive impairment. Consequently, the number of suitable DYNOPTA studies that could be used to address these issues was reduced to include only the ALSA and BMES. Additionally, the final two results chapters to be presented in this thesis explore links between hearing thresholds and processing speed. These two chapters refer solely to the ALSA study as this was the only sample to obtain both these measures.

Thus, the substantive focus of this thesis on hearing loss and cognitive decline is a shift from my original research proposal which initially placed greater emphasis on harmonisation methodology and issues pertaining to multi-study analysis. Nevertheless, substantial work was performed during my candidature on data management, harmonising and pooling the nine contributing DYNOPTA studies. This work has been documented in a number of peer-reviewed publications (Anstey, Bielak, et al., 2011a; Kiely, Gopinath, Mitchell, Browning, et al., 2012; Kiely et al., 2011) and published conference abstracts (Kiely & Anstey, 2009a, 2009b). Although this methods section

describes BMES and ALSA samples and measures, some sections of the methods may make reference to other studies to highlight harmonisation issues.

3.1.2 Studies Contributing to the Primary Objectives of this Thesis

A description of the pooled ALSA and BMES sample by wave is presented in Table 3.2. At baseline there were 4421 participants (46% Men) with an overall mean age of 74.2 (SD = 8.8, range: 50-103). There was considerable drop off in available sample size between the first and second follow-up waves. This was due to the BMES sample only providing hearing and cognitive data at the initial two waves, and a six-year interval in ALSA where sensory and cognitive functioning was not assessed.

3.1.2.1 The Australian Longitudinal Study of Ageing (ALSA)

ALSA (Luszcz et al., 2007) drew a random sample of adults aged 70 years and older from the electoral role for the Adelaide metropolitan area of South Australia in 1992. The sample was stratified by 5-year age groups, sex and government area. In anticipation of lower response rates, ALSA included oversampling of males aged 85 years and older. There were 3263 subjects identified in the primary sample frame, which elicited a 55% response rate. Spouses aged 65 years and older, or adults aged over 70 who were cohabiting with a respondent were also recruited, resulting in a total baseline sample size of 2087 participants. Data collection pertinent to the aims of this thesis occurred within ALSA at wave 1 (1992), wave 3 (1994) wave 6 (2000-2001) and wave 7 (2003-2004).

Information was collected in three formats, a personal home based interview, self-completion questionnaire and clinical assessment. Both the personal interview and clinical assessment contributed to data used in this thesis, and each was conducted by trained personnel. All participants completed the home based interview, which covered

Table 3.2 Participation rates, time intervals and age range for each wave in the pooled ALSA and BMES sample.

Wave	Sample			Years since Baseline		Age		
	Participated in wave	Lost to Attrition	Deceased (cumulative)	Mean	(SD)	Mean	(SD)	Range
Men								
Baseline	2055	387	196	0.0	(0.0)	74.2	(8.8)	50-101
1st Follow Up	1618	542	478	3.8	(1.8)	76.8	(7.9)	55-100
2nd Follow up	335	164	557	8.1	(0.2)	84.4	(5.2)	75-101
3rd Follow up	183	155	718	11.2	(0.2)	85.7	(4.5)	78-102
Women								
Baseline	2387	556	160	0.0	(0.0)	73.1	(8.9)	53-103
1st Follow Up	2013	660	430	3.6	(1.7)	75.8	(7.9)	59-105
2nd Follow up	456	204	371	8.1	(0.2)	83.0	(5.8)	72-100
3rd Follow up	304	218	509	11.2	(0.2)	84.5	(5.1)	75-102
Overall								
Baseline	4442	943	356	0.0	(0.0)	73.6	(8.9)	50-103
1st Follow Up	3631	1,202	908	3.9	(1.8)	76.2	(7.9)	55-105
2nd Follow up	791	368	928	8.1	(0.2)	83.6	(5.6)	72-101
3rd Follow up	487	373	1,227	11.2	(0.2)	84.9	(4.9)	75-102

Note: BMES only contributed to baseline and 1st follow-up.

socio-demographics, self-reported medical conditions and health, psychological screens of cognition and mental health. The clinical assessment was conducted two weeks after the home based interview and included tests of hearing, vision, processing speed and other tests of physical functioning. At baseline 1611 (77.2%) of participants completed the clinical assessment.

3.1.2.2 The Blue Mountains Eye Study (BMES)

The BMES (Tay et al., 2006) attempted to recruit all adults aged 49 years and older from two post-codes in Blue Mountains region west of Sydney, Australia. Data collection pertinent to the aims of this thesis occurred within BMES at wave 2 (1997-1999) and wave 3 (2002-2004). The original baseline sample comprised 3654 participants and had a response rate of 82.4%. During the 3 year interval between wave 1 and wave 2, 943 participants were lost to attrition and 356 were deceased. For most analyses presented in this thesis, baseline was defined by wave 2 of BMES. When deriving population estimates (Chapters 4 and 5) the wave 2 sample was weighted to reflect the age and sex distribution at baseline. These weights were used to account for attrition and mortality over the first follow-up interval. Information on socio-demographics was obtained by self-completion questionnaire and clinical measures were assessed by trained interviewers.

3.1.3 Other Studies Collecting Cognitive and Sensory Data

3.1.3.1 The Canberra Longitudinal Study of Ageing (CLS)

The CLS drew a random of sample of 896 community dwelling adults aged 70 years and older from the compulsory electoral roll of Australian Capital Territory and Queanbeyan area of Australia in 1990. The initial wave had a response rate of 69%, and participants were followed over 12 years on four occasions. The CLS obtained self-

reported sensory functioning data at all waves and corrected visual acuity at waves 3 and 4. A range of cognitive measures were collected at each wave.

3.1.3.2 The Sydney Older Persons Study (SOPS)

The SOPS sample comprised 730 community dwelling adults aged 75 years and older from the inner-west of Sydney in 1991-1993. Participants were randomly sampled from either the Australian Department of Veterans Affairs (n=327) or the Australian Bureau of Statistics census districts (n=320). Seventeen participants were sampled twice. There was an initial response rate of 82% and 73% for the veteran and non-veteran participants respectively. Self-reported sensory data was collected at waves 1, 2, 4 and 5. A range of cognitive measures were collected at each wave.

3.1.3.3 The Personality and Total Health (PATH) through life Survey

The PATH through life survey (Anstey, Christensen, et al., 2011) is an accelerated cohort design comprising three five-year birth cohorts aged 20-24, 40-44 and 60-64 at baseline. Participants were selected at random from the compulsory electoral rolls for the Australian Capital Territory and Queanbeyan. Only the 60s cohort were included in the DYNOPTA pooled dataset. This cohort had a baseline year of 2001-2002 had an initial response rate of 58.3%.

3.1.4 Harmonisation of Sensory Measures

3.1.4.1 Hearing thresholds

Hearing thresholds were assessed by trained interviewers in both ALSA at interview years 1, 3, 6 and 7, and in BMES at interview years 2 and 3. ALSA used calibrated portable audiometers with standard headphones. BMES tested hearing in a sound proof booth with calibrated audiometer. The use of portable audiometers

increases the risk of external sound intrusion during testing. It might therefore be expected that ALSA participants will have slightly higher estimated hearing thresholds compared to matched BMES participants who were tested in a sound proof booth. Both studies assessed thresholds in each ear at frequencies of 0.5, 1, 2, 3, 4, 6 and 8 kHz. The primary hearing measure used in this thesis was a pure tone average (PTA) of hearing thresholds for frequencies of 0.5, 1, 2 and 4 kHz in the better ear. The unit of measurement for hearing thresholds is decibel hearing level (dB HL). Table 3.3 shows the pooled sample measurement properties of PTA by each wave and Figure 3.2 present box plots. On each measurement occasion there was slight positive skew with outliers predominately found for higher hearing thresholds.

3.1.4.2 Hearing Loss

Throughout this thesis a distinction is made between hearing thresholds and Hearing Loss. The term hearing thresholds refers to peripheral hearing function on a linear scale, while the term Hearing Loss refers to impaired hearing levels as defined by ranges of hearing function. In line with standard classifications of Hearing Loss (World Health Organization, 1999), ranges of PTA were used to define a degree of audiometric hearing impairment. Specifically, normal hearing was defined as PTA $\leq$ 25 dB HL, any hearing loss as PTA > 25 dB HL, mild Hearing Loss as PTA > 25 db HL and PTA $\leq$ 40 dB HL, and moderate to severe Hearing Loss as PTA > 40 dB HL. Note that alternate definitions of Hearing Loss are occasionally used in the USA. At all waves there were greater numbers of participants with some degree of Hearing Loss (PTA > 25 dB HL) than participants with normal levels of hearing (PTA $\leq$ 25 dB HL).

Table 3.3 Descriptive data for outcome sensory and cognitive measures by measurement occasion.

	n	Impaired	M	(SD)	Range	Skew	Kurtosis
Hearing Thresholds[a]							
Baseline	3523	52%	28.1	(15.1)	1.3 - 116.3	0.9	4.6
1st Follow Up	3005	57%	30.1	(15.5)	1.3 - 112.5	0.8	4.1
2nd Follow up	525	72%	37.0	(14.3)	6.3 - 108.8	0.9	5.2
3rd Follow up	391	81%	38.6	(15.3)	8.8 - 90.0	0.9	4.2
Visual Acuity[b]							
Baseline	3573	16%	0.70	(0.26)	0.1 - 1.23	-0.13	2.35
1st Follow Up	2913	16%	0.70	(0.26)	0.1 - 1.23	-0.17	2.16
2nd Follow up	416	25%	0.58	(0.24)	0.1 - 1.0	0.20	2.43
3rd Follow up	264	20%	0.63	(0.24)	0.1 - 1.0	0.22	2.21
Mini Mental State Examination (MMSE)[c]							
Baseline	4298	8%	27.7	(2.8)	2.0 - 30	-2.3	11.2
1st Follow Up	3239	7%	27.8	(2.8)	2.0 - 30	-2.4	11.8
2nd Follow up	652	8%	27.6	(2.5)	16.0 - 30	-1.7	6.4
3rd Follow up	407	51%	23.1	(2.0)	9.0 - 29	-1.7	11.2
Digit Symbol Substitution (DSS)[d]							
Baseline	1243		29.2	(11.1)	0.0 - 72.0	0.2	2.9
1st Follow Up	1201		29.8	(11.3)	2.0 - 72.0	0.2	3.0
2nd Follow up	465		29.8	(10.4)	1.0 - 64.0	0,2	3.1
3rd Follow up	357		28.1	(10.7)	0.0 - 67.0	0.1	3.3

[a] Higher values indicate poorer hearing thresholds (dB HL); values above 25 db HL were defined as hearing impairment.

[b] Higher values indicate superior visual acuity; values below 0.5 were defined as visual impairment.

[c] Higher MMSE scores indicate better cognitive function; values below 24 were defined as cognitive impairment

[d] Higher Digit Symbol Substitution scores indicate better processing speed. Impairment level not defined.

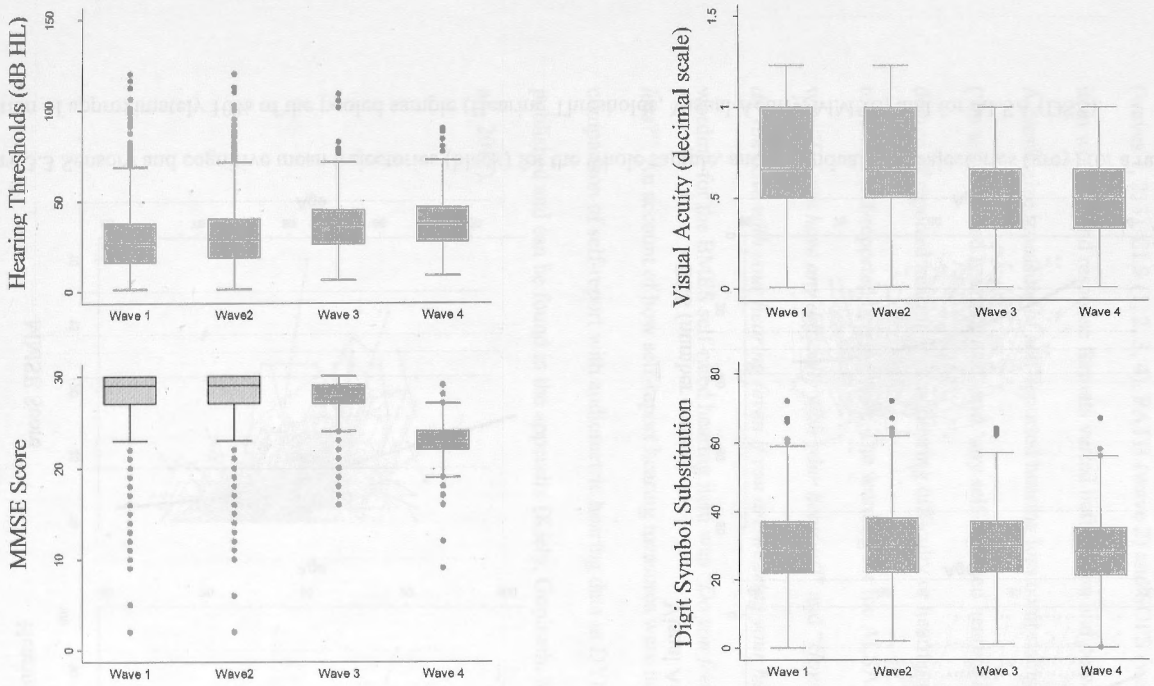


Figure 3.2 Box plots of sensory and cognitive outcome variables by measurement occasion.

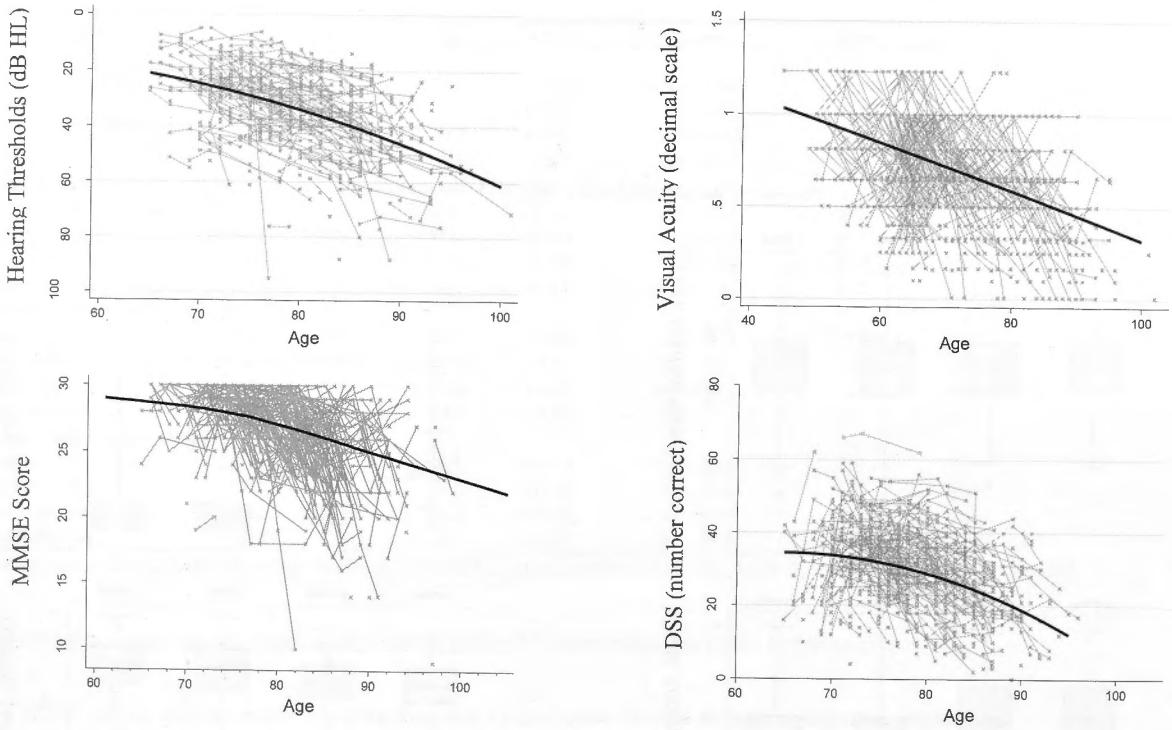


Figure 3.3 Sensory and cognitive mean trajectories (black) for the whole sample, and individual raw trajectories (grey) for a random selection of approximately 10% of the pooled sample (Hearing Thresholds, Visual Acuity, MMSE) and for ALSA (DSS).

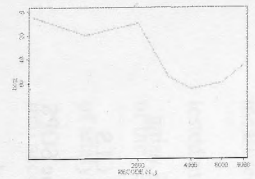
3.1.4.3 Self-reported hearing difficulties

Self-reported hearing loss was obtained by ALSA (waves 1, 3, 6, 7), BMES (waves 1, 2, 3), CLS (1, 2, 3, 4), PATH (wave 2) and SOPS (waves 1, 2, 4, 5). Original item wording and response formats varied both within and between studies (Table 3.4). A harmonized measure of self-reported hearing loss comprising two levels was created ('no self-reported hearing loss', and 'any self-reported hearing loss'), whereby any degree of reported hearing losses, hearing difficulty or hearing problems were recoded to reflect self-reported hearing loss. The wording for the ALSA self-rated hearing item was "*Do you have any difficulty with your hearing*?" and "*How much difficulty, if any, do you have with your hearing (even if you are wearing your hearing aid)?*". The wording for the BMES self-rated hearing item was "*Do you feel you have hearing loss*?". An account of how self-report hearing measures were harmonized, and a comparison of self-report with audiometric hearing data in DYNOPTA has been published and can be found in the appendix (Kiely, Gopinath, Mitchell, Browning, et al., 2012).

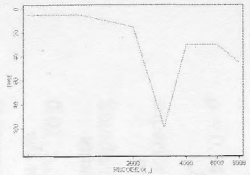
Table 3.4 Self-reported hearing items harmonized in DYNOPTA

Study	Wave	Self-reported hearing Item	Normal hearing	Hearing impairment		
ALSA	1	Do you have any difficulty with your hearing?*	No	Yes		
	1	If yes, how much difficulty do you have with your hearing?		Slight	Moderate	Severe
	3, 6, 7	How much difficulty, if any, do you have with your hearing (even if you are wearing your hearing aid)?*	None	Slight Difficulty	Moderate difficulty	Great difficulty
BMES	1	Have you ever had a problem with your hearing*	No	Yes		
	1	Assessment of hearing problem		Mild	Moderate	Severe
	2, 3	Do you feel you have hearing loss*	No	Yes		
	3	Do you have difficulties with your hearing?	No	Sometimes	Yes	
CLS	1, 2, 3, 4	Would you say you're hearing (with a hearing aid) is generally good, fair or poor?	good	fair	poor	
PATH	2	How you would rate your hearing on the following scale?	Adequate for all purposes	Slight inconvenience	Definite inconvenience	Definite handicap
SOPS	1, 2	Do you have any loss of hearing	No	Yes		

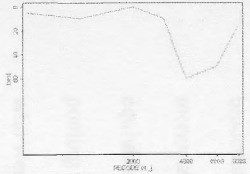
* Items used in harmonised self-reported hearing variable for ALSA and BMES.



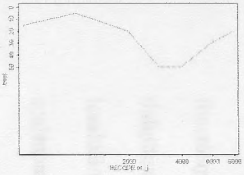
71 year old man reporting 5 years or more of occupational noise exposure, PTA = 25.0 dB HL



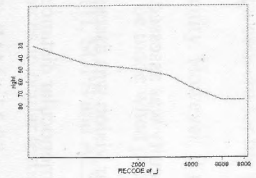
71 year old man reporting 5 years or more of occupational noise exposure, PTA = 13.8 dB HL



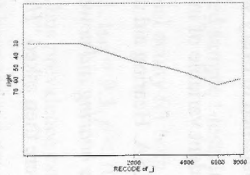
72 year old man reporting 5 years or more of occupational noise exposure, PTA = 18.8 dB HL



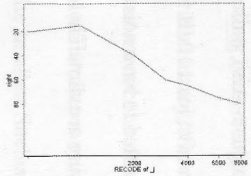
69 year old woman reporting 5 years or more of occupational noise exposure, PTA = 22.5 dB HL



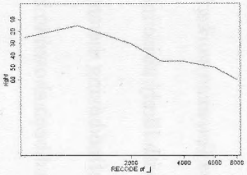
81 year old woman reporting no occupational noise exposure, PTA = 43.5 dB HL



73 year old man woman reporting no occupational noise exposure, PTA = 40.0 dB HL



73 year old male reporting 5 years or more of occupational noise exposure, PTA = 33.7 dB HL



71 year old woman reporting no occupational noise exposure, PTA = 28.7 dB HL

Figure 3.4 Eight randomly selected audiograms and participant characteristics. The top row shows audiograms that were identified to have noise notches by Coles et al. (2000) criteria. The bottom row shows audiograms that were not identified to have noise notches.

3.1.4.4 *Noise damage and noise exposure*

Information on workplace noise exposure was collected in ALSA with the question "*have you ever worked in a noisy environment where you had to shout to be heard*", and in BMES with the question "*have you ever worked in a noisy industry or noisy farm environment*". To identify cases with likely noise induced hearing loss, high frequency audiometric noise notches were defined using the criteria described by Coles, Lutman and Buffin (2000). These criteria have been shown to have strong agreement with expert consensus (Rabinowitz et al., 2006). In audiograms where the y-axis has been reversed (higher thresholds are closer to the x-axis), noise notches are characterised by a downward bulge or trough of at least 10dB for frequency tones around 4kHz. Examples of audiograms identified to have noise notches, along with participant age, sex and reported noise exposure are shown in Figure 3.4.

3.1.4.5 *Hearing aids*

Information on hearing aid use was obtained in every wave of ALSA and BMES. Hearing aid use is the main independent variable of interest in Chapter 8, however this chapter only uses data from ALSA. Hearing aid use was identified in ALSA by the question "*Do you usually use a hearing aid nowadays?*" if participants confirmed they did use a hearing aid, they were then asked "*Has this only been in the last 12 months*".

3.1.4.6 *Visual acuity*

Corrected distance monocular visual acuity was assessed by ALSA, BMES using logMAR charts. Corrected distance binocular vision was assessed by PATH and CLS with Snellen charts. The CLS also assessed monocular distance visual acuity. The SOPS only tested binocular vision acuity for a ratio of 0.3 logMAR (0.5 units in decimal scaling). The harmonised visual acuity variable comprised visual acuity in the better eye

where monocular data were available and binocular visual acuity where monocular data were not available. Although there remains debate on how to classify ranges of visual impairment (e.g. Colenbrander, 2002), robust cross population harmonisation of visual functioning data is aided by internationally adopted standards. In all analyses within this thesis vision impairment is defined as visual acuity greater than 0.3 logMAR (or less than 0.5 in decimal notation) in the better eye. Visual acuity in the better eye was scaled on a logMAR metric when analysed as a linear covariate (Holladay, 1997).

The benefits of binocular vision over monocular vision via the process of binocular summation have long been recognised (Home, 1978). As CLS assessed both monocular and binocular visual acuity, there was an opportunity to investigate the equivalence of binocular and monocular data and quantify the extent to which directly pooling binocular and monocular data may introduce bias. After excluding participants who were unable to read the smallest Snellen line correctly, the correlations between monocular and binocular vision within CLS were moderate (left eye $\rho = 0.53$, right eye $\rho = 0.54$) (Table 3.5). Paired sample t-tests indicated that binocular visual acuity was superior to monocular visual acuity for both the left eye (Mean Difference (MD) = 0.10, Standard Error (SE) = 0.01, $t_{343} = 9.3, p < .01$) and the right eye (MD = 0.10, SE = 0.01, $t_{343} = 9.3, p < .01$). Thus prevalence estimates of visual acuity based on binocular measurements are likely to be more conservative than prevalence based on monocular measurements. This is not a concern for the present thesis as both ALSA and BMES tested monocular visual acuity. However, it is mentioned here to again point out some of the decisions that must be made when pooling data. For example, the interpretation of analyses of pooled PATH, SOPS and ALSA visual acuity data would have to take into account study differences in ocular testing. Potential solutions could include modelling a study effect, or using CLS data to statistically adjust using latent variable techniques.

Table 3.5 Descriptive statistics for monocular and binocular corrected visual acuity for CLS wave 3.

	Descriptives			Variance, covariance and correlation matrix		
	n	M	SD	Left eye	Right eye	Binocular
Left eye	344	0.41	0.20	0.04	0.54	0.53
Right eye	344	0.41	0.19	0.02	0.04	0.54
Binocular	347	0.51	0.21	0.02	0.02	0.05

Correlations are shown above the diagonal, variances on the diagonal and covariance below the diagonal. Participants unable to read the smallest Snellen line correctly were defined as functionally blind and excluded from analysis.

3.1.5 Harmonisation of Cognitive Measures

3.1.5.1 Mini-Mental State Examination (MMSE)

The Mini Mental State Examination (MMSE: Folstein et al., 1975a) is a screen for cognitive impairment that is often used in clinical settings and epidemiological surveys. It includes the domains of orientation, registration, recall, working memory, language, and following instructions. Meta-analyses have identified that the main value of MMSE in population based samples is in ruling out dementia cases (Mitchell, 2009). MMSE was the most common cognitive measure shared by the contributing DYNOPTA studies and was the only cognitive measure in the BMES. Possible cognitive impairment was defined by an MMSE score of 23 or less. The MMSE was validated against clinical diagnoses of dementia in DYNOPTA (though not in the ALSA and BMES sub-samples), and was shown to have sensitivity of 93% and specificity of 70% (Anstey, Burns, et al., 2010).

There are well recognised limitations of the MMSE as a screen of cognitive impairment or measure of global cognitive function. Firstly, although it taps into a number of cognitive domains, it does so at a basic level and does not assess abstract reasoning, executive function or visual perception (Aihong & Jianping, 2008). As evident in Figure 3.3 and 3.4, the MMSE typically exhibits strong ceiling effects and non-linear characteristics (Proust-Lima, Dartigues, & Jacqmin-Gadda, 2011). Its common use in a number of research, clinical and care settings means older adults may be regularly exposed to MMSE items. Consequently, the MMSE may be susceptible to practice effects. Though this may enhance the value of the MMSE in identifying possible dementia cases, it is a poor index of normal cognitive function among healthy adults and even its effectiveness in identifying mild cognitive impairment has been questioned (Aihong & Jianping, 2008; Mitchell, 2009). Finally, longitudinal analyses have shown that there is variation in the rate of decline in performance across the MMSE items (Tinklenberg et al., 1990).

Differential item functioning in relation to education level has been reported in the MMSE (Crane et al., 2006; Jones & Gallo, 2002). Such education bias has led to a number of researchers advocating and using education specific cut-points when defining cognitive impairment with the MMSE (Kahle-Wrobleski, Corrada, Li, & Kawas, 2007; Matthews, Jagger, Miller, & Brayne, 2009). However, education may be a risk factor for cognitive impairment (as proposed by the cognitive reserve hypothesis) or a good proxy for lifestyle behaviours that are also risk factors for cognitive impairment, so education differences may reflect true differences that should not be characterised as bias (Jorm, Scott, Henderson, & Kay, 1988; Kraemer, Moritz, & Yesavage, 1998). Jorm and colleagues (1998) have reported that there was no MMSE educational bias for an Australian community based sample when validated against ADL criteria. For these reasons, a single cut-point was used and analyses adjusted for education as a covariate.

This also avoids effectively adjusting for education twice, when defining cognitive impairment, and when the conducting analyses.

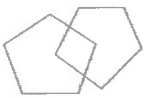
Despite its ubiquitous use, there were study differences in the coding of missing MMSE data within DYNOPTA. Whereas some studies used generic missing data codes, other specifically coded the reason for non-response. For example, if a participant was unable to attempt an item due to visual impairment[2], this was explicitly coded in SOPS and BMES. It was also apparent that some item level missing data was actually indicative of an incorrect response. These issues were addressed when harmonising the MMSE using imputation techniques (Burns et al., 2011).

3.1.5.2 Processing speed

Speed of information processing is an elementary cognitive resource necessary for efficient functioning. Dual process models of cognitive development place processing speed in the class of fluid intelligence abilities (Horn & Cattell, 1967), also sometimes referred to as cognitive mechanics (Baltes, 1987). Fluid abilities are basic cognitive operations that underpin abstraction, classification, reasoning and general problem solving. A distinguishing characteristic of this set of abilities is their decline with age. There is some debate concerning when in the lifespan processing speed begins to decline (Salthouse, 2009), but certainly by midlife slowing of processing speed can be assumed to have commenced and by late-life decline is clearly evident (Figure 3.3).

Processing speed is a key construct of cognitive ageing (Birren & Fisher, 1995) and has been central to a number of influential theories including the generalized

[2] For example, the item "Copy this design", which requires participants to draw two intersecting pentagrams.



slowing hypothesis (Cerella, 1994), the processing speed hypothesis (Salthouse, 1996) and common cause hypothesis (Lindenberger & Baltes, 1994). Processing speed has consistently been shown to explain substantial portions of age-related variance in a range of cognitive functions (Salthouse, 1991; Salthouse, 1993; Salthouse, 2000). In light of this mediating role, processing speed has been has afforded special status as a cognitive primitive (Verhaeghen, Steitz, Sliwinski, & Cerella, 2003) which may act as a functional driver of changes in memory (Luszcz & Bryan, 1999) and other fluid abilities (Rabbitt et al., 2007).

Processing speed measures can be grouped into four broad types; decision speed, psychomotor speed, psychophysical speed and perceptual speed (Salthouse, 2000). Perceptual speed tasks require participants to perform multiple search, comparison and substitution operations within a set time limit. The Digit Symbol Substitution (DSS) (Wechsler, 1981) test is one example of a perceptual speed task that has a long tradition in cognitive ageing research (Erber, 1976; MacDonald, Hultsch, Strauss, & Dixon, 2003; Salthouse, 1992) and was used as a measure of processing speed in ALSA. The DSS requires participants to match as many numerical digits paired with nine symbols as possible in 90 seconds (Figure 3.5) Test-retest reliability was .79 (Luszcz, Bryan, & Kent, 1997). An advantage of processing measures over other cognitive measures is their wide number of data points, which increases precision and captures a broad range of functioning in in normal healthy samples. The DSS is an outcome measure in Chapters 8 and 9, because the BMES did not collect a comparable measure of processing speed it was excluded from the analyses presented in these chapters.

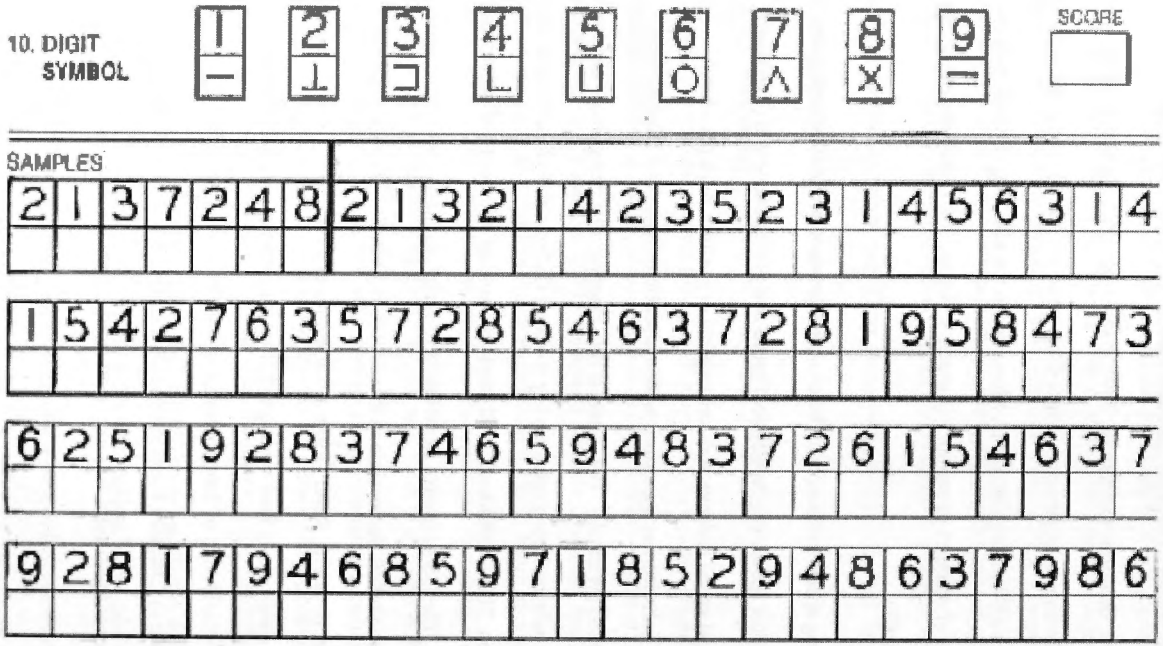


Figure 3.5 The Digit Symbol Substitution (DSS) Test (Wechsler, 1981) was used as a measure of processing speed in the ALSA. Participants are required to match as many symbols with their paired digit in 90 seconds. Similar processing speed tasks were collected in the CLS and PATH but do not form part of this thesis.

Other processing speed measures available in the DYNOPTA dataset were collected in PATH (Symbol Digit Modalities Task) and CLS (Symbol Letter Modalities Task). Although these samples will not be included in the present thesis, it is interesting to point out subtle differences between these measures that would have to be taken into account when interpreting analyses of pooled PATH, CLS, and ALSA processing speed data. The speeded task in CLS had a verbal response modality, whereas the DSS in ALSA required written responses. It could be expected that as older adults become frail with age, performance on a speeded task that requires written responses will decline at a faster rate than performance on a verbal task.

3.1.6 Covariates

Mortality data was obtained by linkage with the National Death Index. Within DYNOPTA, the censoring date for ALSA was January 2008, and for BMES was February 2007. Smoking status was coded as never smokers, former smokers and current smokers. Self-report of clinician diagnosed medical conditions included diabetes, hypertension, cardiovascular disease, history of stroke, and cancer. The CES-D was used as a measure of depression in ALSA (Radloff, 1977).

There were two education variables harmonized in DYNOPTA, 'Age Left School', and 'Highest Qualification Attained'. Although age left school was collected by ALSA, it was not collected by BMES. Both studies did however collect data on qualifications (Secondary Schooling Only, Post-secondary non-tertiary and Tertiary). A measure of *highest qualification attained* was harmonized across all nine studies. Although some studies finely coded more than 300 individual qualification types, these were recoded to a coarser level comprising three response categories which were common to all studies ('Tertiary', 'Post-secondary but non tertiary', 'Secondary only'). There are a couple of limitations to acknowledge with this harmonized education

variable. Firstly, the need to collapse response categories to create a common scale across all studies resulted in a loss of information and reduced variability in the data. Secondly, because BMES asked '*have you obtained a qualification since leaving school*', participants from this study who reported no qualifications were assumed to have secondary schooling as their highest level of educational attainment. Therefore, it is not possible to distinguish between different levels of secondary schooling.

Career Occupation was harmonized from questions that asked about participants' main or current occupation during their working life. Seven studies coded career occupation according to the either the first (MELSHA, ALSWH) or second (ALSA, AusDiab, BMES, HILDA, PATH) edition of the Australian Standard Classification of Occupation (ASCO I and II). SOPS coded 16 occupation classifications according to the ANU III taxonomy, while the CLS coded 6 occupation types that were derived from the ANU III (Broom, Duncan-Jones, Jones, & McDonnell, 1977; Broom, Duncan-Jones, Lancaster Jones, & McDonnell, 1977; Quine, 1986). A harmonized measure of career occupation with 4 occupation classifications was created, this equated to collapsed major group (single digit code) categories from the ASCO II ('Managers and Professionals', 'Clerical and Associate Professional', 'Tradespersons' and 'Sales, Service, Production, Transport and Labourers') (DYNOPTA Working Party, 2008a, 2008b).

3.1.7 Statistical Analyses

A range of longitudinal statistical models will be used throughout this thesis, including interpolated Markov chains, linear mixed models, cox regression, joint parameter growth curve-survival models and bivariate dual change score models. Each analytic technique will be described in detail within the relevant chapter. Chapters 4 and 5 focus on sensory loss and cognitive impairment so will use categorical measures of

hearing, vision and cognition. Chapters 7, 8 and 9 focus on hearing and cognitive decline and so use continuous measures of hearing and cognition (processing speed). A glossary of statistical terms can be found on page xxi.

3.1.8 Optimal Scaling of Time

Determining the appropriate orientation of time is an important step when conducting analysis of longitudinal data, for both event-history models (Michael, Martin, & Ralph, 2007) and growth curve models (Gerstorf, Ram, Röcke, Lindenberger, & Smith, 2008). Due to variability in time intervals between waves in DYNOPTA, it is not appropriate to model change discreetly by wave number. Unless otherwise stated[3], time in study (years) was used as the metric of time and adjusted for baseline age, which is consistent with previous recommendations of modelling time with longitudinal data with a broad age range (Morrell, Brant, & Ferrucci, 2009).

[3] In chapter 5 age was used as the time metric

CHAPTER 4: Prevalence, Incidence and Risk Factors for Hearing Loss and its Co-morbidity with Vision Loss and Cognitive Impairment

Synopsis

The literature review in Chapter 1 reported that hearing loss is one of the most prevalent chronic conditions found in older adults. Sensory loss and neurological impairment are two leading contributors to years lived with disability among older adults, yet despite evidence of links between these two domains population estimates of their co-morbidity have received little attention. Further there is a perception that self-report sensory data is a reliable proxy for clinical measures and in some contexts may provide accurate prevalence estimates. This descriptive chapter focuses on hearing loss and its co-morbidity with vision loss and cognitive impairment at the population level, providing estimates of prevalence, and incidence. Comparisons of functional and perceived health status in relation to hearing will also be made.

4.1 Background

There has been little documentation of prevalence or incidence of co-occurring sensory and cognitive impairment. In fact, there are instances where both conditions have been overlooked and excluded from studies into the level and cost of co-morbidities. For example, a recent AIHW report on co-morbidities between mental disorders and physical conditions in Australia using National Survey of Mental Health and Wellbeing data (Australian Institute of Health and Welfare, 2011) did not include cognitive impairment as a mental disorder nor did it include sensory loss as a physical condition. While this may be due to the report's focus on the full adult lifespan, given that the oldest old (ages 80 years and older) is the fastest growing segment of the Australian population it is important, from both policy and clinical perspectives, to understand how these age-related conditions co-occur.

The gold standard method for measuring Hearing Loss is pure-tone audiometry. However, because the costs and logistics involved in conducting audiometry assessments are prohibitive for many epidemiological surveys, self-report measures are often used instead (Shield, 2006). These self-reported measures of hearing loss have previously been thought to be reliable and predictive of measured hearing loss whilst also providing an ecologically valid measure of perceived hearing difficulties (Nondahl et al., 1998).

The purpose of this chapter is largely descriptive. It will use clinical measures collected longitudinally to provide population estimates of prevalence and incidence of age-related hearing loss, dual sensory loss (DSL) and hearing loss co-morbid with cognitive impairment. Use of self-report measures will also be evaluated by comparing population estimates based on self-rated hearing difficulty with clinically defined audiometric Hearing Loss.

4.2 Methods

4.2.1 Participants

This study used participants who completed hearing, vision and cognitive assessments from the ALSA waves 1, 3, 6 and 7 and BMES waves 2 and 3 (Table 4.1). The BMES wave 2 sample was weighted to reflect baseline characteristics.

Table 4.1 Time schedule for ALSA and BMES.

Measurement Occasion	ALSA			BMES		
	start	finish	Hearing data collected	start	finish	Hearing data collected
1	Sep-92	Feb-93	Yes	Jan-92	Dec-93	.
2	Jan-93	Apr-94	.	Jan-97	Feb-00	Yes
3	Jan-94	Dec-95	Yes	May-01	Dec-04	Yes
4	Jan-95	Dec-96	.	.	.	.
5	Feb-98	Apr-98	.	.	.	.
6	Sep-00	Dec-01	Yes	.	.	.
7	Sep-03	Apr-04	Yes	.	.	.

4.2.2 Measures

Hearing loss was defined by standard ranges of PTA in the better ear (PTA>25 dB = Any hearing loss; PTA > 25 dB & PTA ≤ 40 dB = Mild hearing loss; PTA > 40 dB = Moderate hearing loss). Vision loss was defined by corrected distance visual acuity greater than 0.3 logMAR in the better eye. Cognitive impairment was defined by a MMSE score of 23 or lower. A harmonised dichotomous measure of self-reported hearing difficulty was used as an indicator of perceived hearing loss.

4.2.3 Analyses

Prevalence rates for audiometric hearing loss, self-reported hearing loss and hearing loss co-morbid with either cognitive impairment or vision loss were estimated for the pooled baseline ALSA sample and wave 2 BMES sample stratified by sex and 5

year age groups. BMES was weighted to reflect the baseline sample. Multinomial logistic regression was used to test for sex differences in prevalence of each condition.

Sample averaged audiograms for men and women aged 65 and 85 were produced by graphing the estimated thresholds for frequencies of 0.5-8kHz from regression models. The regression model included the main effects for age (mean centred) and sex as well as their interaction term (see equation 1). This was repeated for cognitive impairment and self-reported hearing loss.

Equation 1: Estimated threshold = intercept + b_1*age + b_2*sex + b_3*age*sex

Incident rates were estimated for mild hearing loss, moderate hearing loss, dual sensory loss, and hearing loss co-occurring with cognitive impairment. To account for study differences in time intervals, annualized incidence rates per 1,000 person-years in the pooled sample were calculated using all available waves of data. Age and sex specific incidence rates were calculated as the number of incident cases divided by the number of person-years at risk. Due to small numbers of incident cases, broad age groups spanning 15 years were used. Participants were no longer considered at risk after the first recorded occurrence of either 1) completion of the study follow-up period, 2) incident DSL, 3) attrition, or 4) death. Because the data were interval censored, the midpoint of the time interval was imputed as the time of incident DSL for new cases. Despite its limitations, this is a common approach to estimating incidence rates when the precise time of onset is unknown (Fratiglioni et al., 2000)

4.3 Results

4.3.1 Prevalence of Hearing Loss and its Co-morbidity with Vision Loss and Cognitive Impairment

Age and sex prevalence of mild and moderate-to-severe Hearing Loss is presented in Table 4.2. Results are reported with a focus on the oldest-old cohort of adults aged 85 years or older. Adults in this cohort were more likely to have a moderate to severe degree of hearing loss than mild hearing loss. Further, almost one in nine adults in this age cohort had at least a mild degree of hearing impairment. In contrast with younger cohorts, there also appeared to be no sex differences in prevalence of hearing loss among the oldest-old. This was confirmed by age and sex adjusted multinomial logistic regression analyses conducted separately for two age ranges. When the baseline sample was restricted to adults aged 85 years or older (n = 349, 53.6% men) women were not at significantly reduced risk of mild Hearing Loss (Odds Ratio (OR) = 1.3, 95% CI = 0.67, 2.56) or moderate Hearing Loss (OR = 0.98, 95% CI = 0.61, 1.58) relative to men. This was in contrast to the younger cohort of adults (n = 3177, 45.5% men), for whom women were at reduced risk of mild Hearing Loss (OR =0.69, 95% CI = 0.58, 0.82) and moderate Hearing Loss (OR = 0.51, 95% CI = 0.41, 0.64) relative to men.

4.3.2 Comparison of Prevalence Derived from Audiometric Hearing Loss and Self-report Hearing Difficulty

An evaluation of population estimates derived from self-reported hearing difficulty compared to audiometric measures based on the findings from this section has been published as part of this doctoral work (Kiely, Gopinath, Mitchell, Browning, et al., 2012). Overall prevalence rates derived from self-reported hearing loss was 48.5% (95% CI = 46.8, 50.1) which only slightly underestimated true overall prevalence

Table 4.2 Pooled sample prevalence of hearing loss by age and sex (n = 3,526)

	Mild Hearing Loss		Moderate Hearing Loss		Self-Reported Hearing Difficulty	
Age	**%**	**[95% C.I.]**	**%**	**[95% C.I.]**	**%**	**[95% C.I.]**
			Men			
55-59	7.6	[4.0, 13.9]	2.5	[0.8, 7.5]	44.7	[35.9, 53.9]
60-64	17.8	[12.4, 24.9]	5.5	[2.8, 10.6]	53.5	[45.3, 61.6]
65-69	28.2	[22.1, 35.2]	16.0	[11.4, 22.1]	56.2	[48.8, 63.3]
70-74	39.9	[35.3, 44.6]	13.4	[10.5, 17.0]	50.6	[46.1, 55.1]
75-79	46.1	[40.8, 51.5]	25.0	[20.6, 29.9]	53.6	[48.6, 58.4]
80-84	41.0	[34.9, 47.4]	41.0	[34.9, 47.4]	58.5	[53.0, 63.9]
85+	35.8	[29.3, 43]	52.4	[45.2, 59.5]	69.3	[63.6, 74.5]
			Women			
55-59	9.2	[5.5, 14.9]	3.3	[1.4, 7.6]	33.4	[26.1, 41.6]
60-64	10.2	[6.8, 15.2]	4.9	[2.6, 7.8]	37.6	[31.1, 44.6]
65-69	23.1	[18.9, 28.0]	4.9	[3.0, 8.8]	47.0	[24.5, 70.8]
70-74	36.6	[32.3, 41.2]	10.4	[7.9, 13.5]	34.5	[27.9, 41.7]
75-79	39.4	[34.4, 44.6]	16.2	[12.7, 20.4]	38.9	[31.4, 46.9]
80-84	42.2	[36.0, 48.7]	31.3	[25.6, 37.6]	41.7	[33.7, 50.2]
85+	33.3	[26.5, 40.9]	52.5	[44.8, 60.1]	64.2	[56.6, 71.2]

Mild Hearing Loss: PTA > 25 dB and PTA ≤ 40 dB

Moderate Hearing Loss: PTA > 40 dB

Table 4.3 Prevalence and 95% confidence intervals of vision loss, hearing loss and Dual Sensory Loss stratified by 5 year group and sex (n = 3190)

	Vision Loss Only		Hearing Loss Only		Dual Sensory Loss	
Age	**%**	**[95% C.I.]**	**%**	**[95% C.I.]**	**%**	**[95% C.I.]**
			Men			
55-59	1.7	[0.4, 6.5]	9.3	[5.2, 16.1]		
60-64	2.8	[1.0, 7.1]	22.1	[16.1, 29.6]	1.4	[0.3, 5.4]
65-69	4.6	[2.3, 8.9]	39.4	[32.5, 46.9]	3.4	[1.6, 7.4]
70-74	3.3	[1.9, 5.7]	47.0	[41.9, 52.1]	6.3	[4.2, 9.3]
75-79	3.9	[2.2, 6.9]	57.6	[51.8, 63.2]	13.8	[10.2, 18.3]
80-84	4.3	[2.2, 8.0]	60.7	[53.9, 67.0]	23.2	[18.0, 29.4]
85+	4.8	[2.3, 9.7]	51.0	[43.0, 59.0]	36.1	[28.7, 44.1]
			Women			
55-59	2.0	[0.7, 6.0]	12.0	[7.7, 18.3]	0.7	[0.0, 4.6]
60-64	3.9	[2.0, 7.7]	14.8	[10.5, 20.4]		
65-69	7.8	[5.3, 11.4]	21.6	[17.3, 26.5]	4.3	[2.5, 7.2]
70-74	5.2	[3.4, 7.7]	43.8	[39.1, 48.5]	4.9	[3.2, 7.5]
75-79	8.1	[5.5, 11.7]	46.4	[40.9, 52.0]	9.1	[6.4, 12.9]
80-84	6.9	[4.1, 11.3]	47.8	[41.0, 54.7]	26.1	[20.5, 32.6]
85+	5.8	[2.9, 11.1]	41.7	[33.8, 50.1]	44.6	[36.6, 53.0]

Hearing Loss: PTA > 25 dB

Table 4.4 Prevalence and 95% confidence intervals of probable cognitive impairment (PCI), Hearing Loss (HL) and co-morbid hearing loss with cognitive impairment stratified by 5 year age group and sex (n = 3448).

	PCI Only		Hearing Loss Only		Co-morbid PCI and HL	
Age	**%**	**[95% C.I.]**	**%**	**[95% C.I.]**	**%**	**[95% C.I.]**
			Men			
55-59	0.9	[0.1, 5.8]	9.3	[5.2, 16.1]		
60-64			21.7	[15.7, 29.2]	1.4	[0.4, 5.4]
65-69	2.2	[0.8, 5.8]	43.6	[36.5, 50.9]	0.6	[0.0, 3.9]
70-74	1.7	[0.8, 3.5]	49.5	[44.7, 54.3]	3.6	[2.2, 6.0]
75-79	1.2	[0.5, 3.2]	64.1	[58.8, 69.1]	6.7	[4.4, 10.0]
80-84	1.3	[0.4, 3.9]	71.2	[65.1, 76.7]	10.7	[7.4, 15.4]
85+	2.2	[0.8, 5.7]	69.0	[62.0, 75.3]	19.0	[14.0, 25.4]
			Women			
55-59	1.3	[0.3, 5.0]	10.2	[6.2, 16.2]	0.8	[0.1, 5.3]
60-64	1.3	[0.4, 4.1]	15.5	[11.0, 21.3]		
65-69	0.2	[0.0, 1.3]	29.5	[14.6, 50.5]	1.0	[0.4, 2.5]
70-74	3.6	[1.6, 8.2]	46.8	[38.8, 54.9]	1.7	[0.5, 5.5]
75-79	0.6	[0.2, 2.4]	55.2	[46.0, 64.0]	7.3	[3.4, 14.8]
80-84	1.0	[0.2, 4.7]	59.7	[49.4, 69.1]	11.5	[6.4, 19.9]
85+	2.5	[0.8, 7.6]	69.5	[59.6, 77.9]	19.7	[12.7, 29.2]

Hearing Loss: PTA > 25 dB

estimated from PTA > 25 dB HL of 51.9% (95% CI = 50.2, 53.6). However, the age gradient for self-report items was not as steep as the age gradient for measured hearing impairment (Figure 4.2). The prevalence of self-reported hearing difficulty increased by 4.1% for every five year increase in age. In contrast, prevalence of audiometric hearing loss increased by 13.5% for every five year increase in age. Although prevalence rates based on self-reported items were reasonably accurate for adults aged between 65 and 74 years, prevalence based on self-report data greatly overestimated measured hearing loss prevalence for younger age groups but greatly underestimated prevalence rates for older age groups. For example, 44.7% (95% CI = 35.9, 53.9) of men aged between 55 and 59 reported some degree of hearing loss, whereas only 10.1% (95% CI = 5.8, 16.9) of men in this age-group had average PTA greater than 25 dB HL in the better ear. In contrast, 69.3% (95% CI = 63.6, 74.5) of men aged over 85 years reported some level of hearing difficulty, whereas 88.2% (95% CI = 82.8, 92.1) of men in this age group had average PTA greater than 25 dB HL in the better ear. A similar pattern was observed for women. Prevalence of hearing impairment based on self-report by women aged over 85 years was 64.2% (95% CI = 56.6, 71.2) which was significantly less than prevalence of 85.8% (95% CI = 79.5, 90.4) of women aged over 85 who had average PTA greater than 25 dB HL in the better ear.

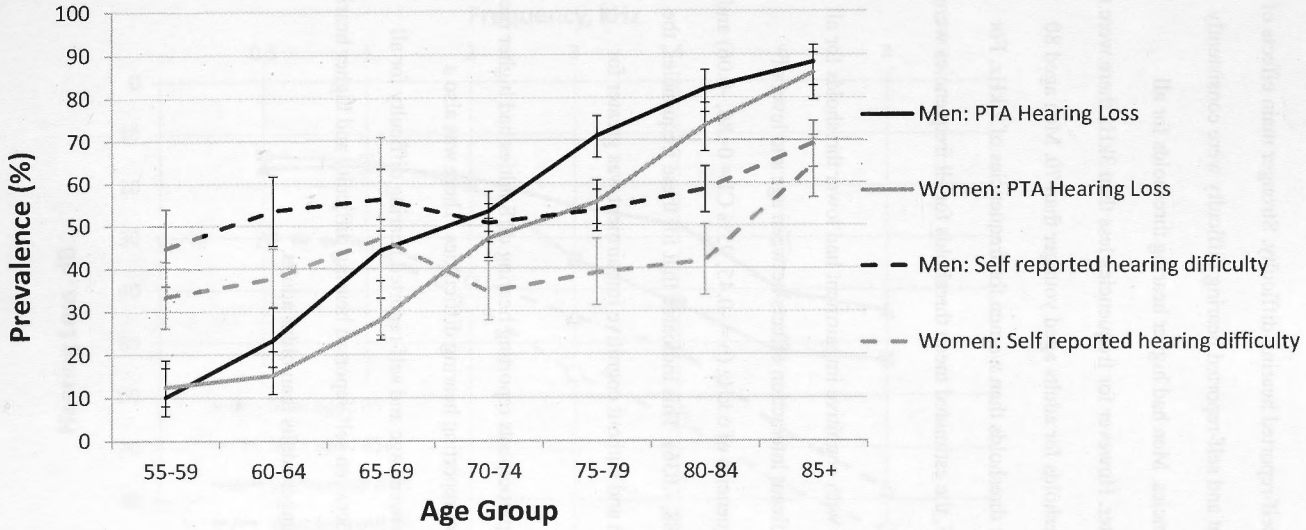


Figure 4.1 Comparison of prevalence derived from either audiometric sensory loss or self-reported hearing difficulty for men and women by 5 year age group. Error bars show 95% confidence intervals.

PTA Hearing loss is defined by PTA > 25 dB HL in the better ear

4.3.3 Mean Audiograms

Figures 4.2-4.4 show the mean audiograms for adults aged 65 and 85 by sex, level of cognitive impairment and self-reported hearing difficulty. Stronger main effects of age, sex, cognitive impairment and self-reported hearing difficulty were consistently found for higher range frequencies. Men had higher hearing thresholds for all frequencies of 3kHz and greater. However for frequencies less than 3kHz there were no sex differences in hearing thresholds for adults aged younger than 70. Men aged 80 years or older did have higher thresholds than women for frequencies of 2 kHz. For both men and women aged 85, the estimated mean thresholds for all frequencies were greater than 25 dB HL

Participants identified with cognitive impairment had lower thresholds for all frequencies. There were significant interaction effects between age and level of cognitive impairment for frequencies of 6 kHz (b = -0.45, 95% CI = 0-.86, -0.06) and 8 kHz (b = -0.46, 95% CI = -0.88, -.036). This indicated that for these frequencies, the difference between those with and without cognitive impairment was greater for younger ages.

Across all frequencies, participants reporting hearing difficulties had higher mean thresholds than participants not reporting hearing difficulties. There was also a significant interaction term between age and self-reported hearing difficulty for all frequencies. The association between self-reported hearing difficulty and higher hearing thresholds was greater for younger adults than older adults.

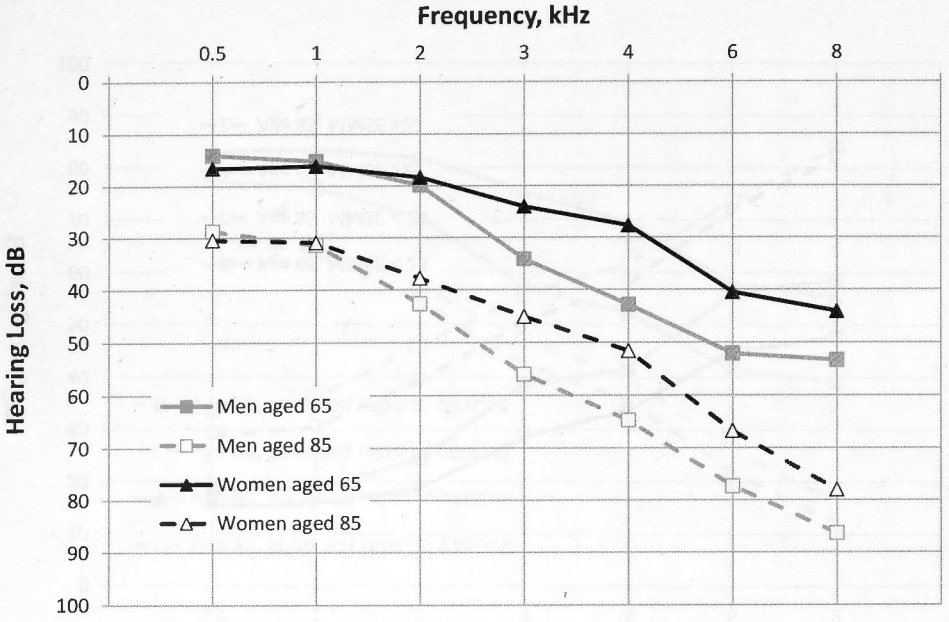


Figure 4.2 Mean audiograms for men and women aged 65 and 85; mean thresholds were estimated from baseline data as a function of age, sex and their interaction.

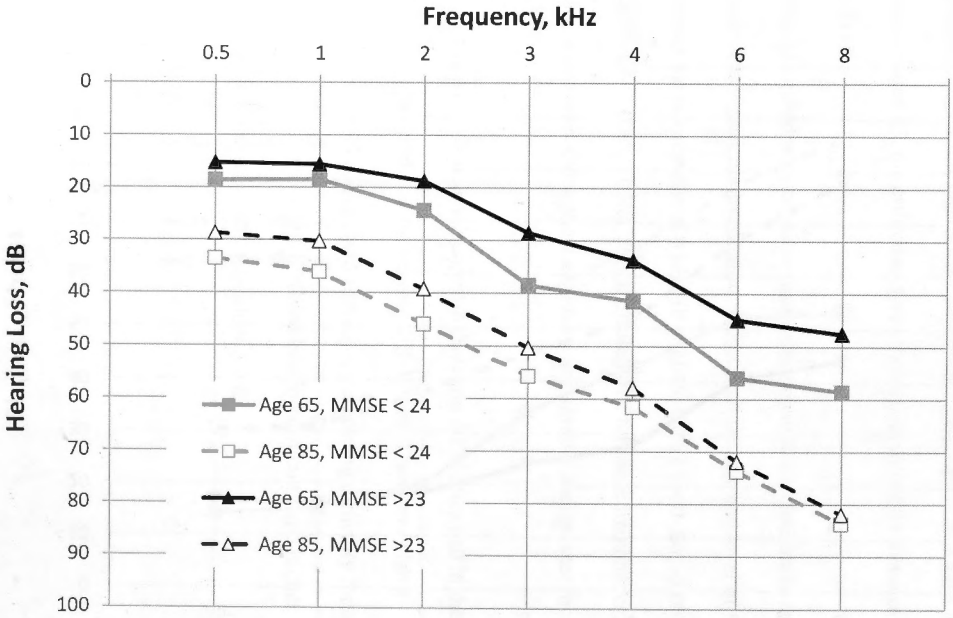


Figure 4.3 Mean audiograms for adults aged 65 and 85 with and without probable cognitive impairment (MMSE < 24), mean thresholds were estimated from baseline data as a function of age, cognitive impairment and their interaction.

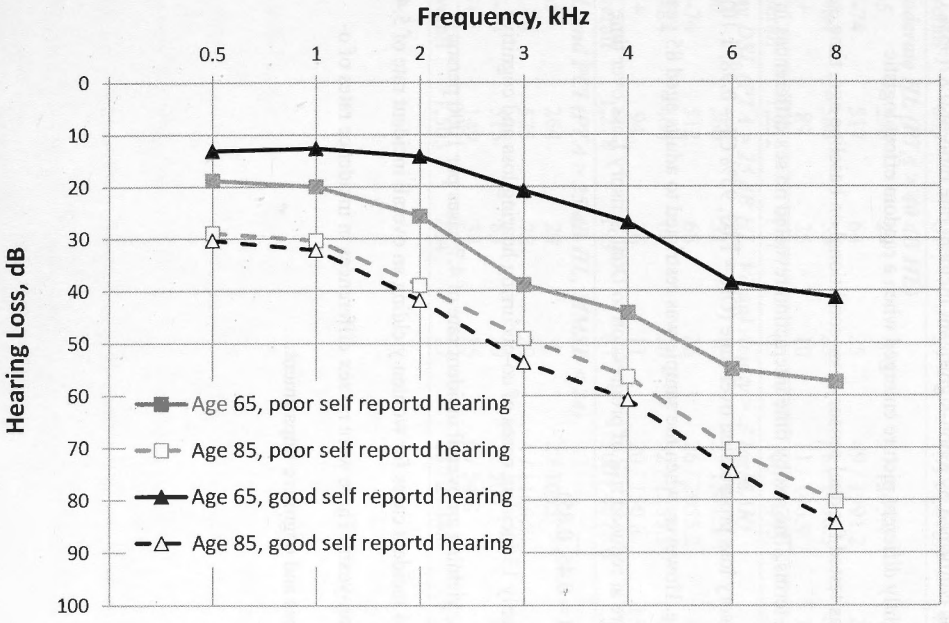


Figure 4.4 Mean audiograms for adults aged 65 and 85 by self-reported hearing difficulty, mean thresholds were estimated from baseline data as a function of age, self-reported hearing difficulty and their interaction.

4.3.4 Incidence Rates

Incidence of hearing loss and its co-morbidity with vision loss or cognitive impairment is given in Table 4.5 for men and Table 4.6 for women. There were 100 incident cases of Dual Sensory Loss for men yielding an overall incidence rate of 21.4 per 1000 person-years, and 121 incident cases for women yielding an overall incidence rate of 18.6 per 1000 person-years. Overlapping 95% confidence intervals for incidence rates of dual sensory loss for men and women indicated that there were no sex differences. This was confirmed by a non-significant incident rate ratio of 0.87(95% CI = 0.66, 1.14). A slightly different picture emerged when a random effect logistic regression model was tested for dual sensory loss conditional on baseline age, time, sex and their interaction terms. The sex by time interaction revealed no sex differences in the odds of dual sensory loss progression over time (OR = 1.03, 95% CI = 0.98, 1.10) for the whole sample. However, when the sample was restricted to adults aged 85 years or older, women were at reduced risk of progression to Dual Sensory Loss over time (OR = 0.62, 95% CI = 0.45, 0.85).

There were only 13 incident cases of co-occurring hearing loss and cognitive impairment for men, yielding an overall incident rate of 4.7 cases per 1000 person-years. There were 24 incident cases for women yielding an overall incident rate of 5.4 cases per 1000 person-years. There were no sex differences in incidence rates of o-occurring hearing loss and cognitive impairment.

Table 4.5 Incident rates for Hearing Loss (HL), Dual Sensory Loss (DSL) and co-occurring hearing and cognitive impairment per 1000 person-years for men.

	Pooled Sample			Pooled Incidence Rate per 1000 Person-Years		
Age at baseline	**Baseline at risk**	**Wave 2 drop out**	**Wave2 deceased**	**Cases / person-years**	**IR**	**[95% CI]**
Mild HL (PTA > 25 dB HL, & PTA <40 dB HL)						
<65	224	23	3	14 / 1022.4	13.7	[8.1, 23.1]
65-74	299	38	15	80 / 1004.0	79.7	[64.0, 99.2]
75-84	138	17	7	52 / 370.7	140.3	[106.9, 184.1]
85+	22	3	4	7 / 43.8	159.7	[76.2, 335.1]
Overall	683	81	29	153 / 2440.9	62.7	[53.5, 73.4]
Moderate HL (PTA > 40 dB HL)						
<65	258	29	3	2 / 1205.4	1.7	[0.4, 6.6]
65-74	517	64	25	49 / 1991.2	24.6	[18.6, 32.6]
75-84	388	53	25	78 / 1130.3	69.0	[55.3, 86.2]
85+	89	25	10	11 / 146.9	74.9	[41.5, 135.2]
Overall	1,252	171	63	140 / 4473.9	31.3	[26.5, 36.9]
Any DSL (PTA > 25 dB HL, Visual Acuity > .3 logMAR)						
<65	266	31	4	2 / 1231.5	1.6	[0.4, 6.5]
65-74	512	68	26	26 / 2055.2	12.7	[8.6, 18.6]
75-84	406	68	26	52 / 1239.1	42.0	[32.0, 55.1]
85+	94	26	11	20 / 150.8	132.6	[85.6, 205.6]
Overall	1,278	193	67	100 / 4676.6	21.4	[17.6, 26]
HL and PCI (PTA > 25 dB HL, MMSE < 24)						
<65	264	29	4	2 / 1055	1.9	[0.5, 7.6]
65-74	575	78	29	4 / 1166.7	3.4	[1.3, 9.1]
75-84	515	83	29	7 / 508.3	13.8	[6.6, 28.9]
85+	149	34	15	0 / 54.8		
Overall	1,503	224	77	13 / 2784.8	4.7	[2.7, 8.0]

Table 4.6 Incident rates for Hearing Loss (HL), Dual Sensory Loss (DSL) and co-occurring hearing and cognitive impairment per 1000 person-years for women.

	Pooled Sample			Pooled Incidence Rate per 1000 Person-Years		
Age at baseline	**Baseline at risk**	**Wave 2 drop out**	**Wave2 deceased**	**Cases / person-years**	**IR**	**[95% CI]**
Mild HL (PTA > 25 dB HL, & PTA <40 dB HL)						
<65	312	27	7	20 / 1454.0	13.8	[8.9, 21.3]
65-74	474	46	6	132 / 1798.3	73.4	[61.9, 87.1]
75-84	216	42	11	78 / 588.8	132.5	[106.1, 165.4]
85+	22	1	1	14 / 52.2	268.2	[158.8, 452.9]
Overall	1,024	116	25	244 / 3893.3	62.7	[55.3, 71.1]
Moderate HL (PTA > 40 dB HL)						
<65	347	33	9	2 / 1642.6	1.2	[0.3, 4.9]
65-74	715	62	9	79 / 3112.4	25.4	[20.4, 31.6]
75-84	451	75	25	75 / 1494.5	50.2	[40.0, 62.9]
85+	76	25	6	10 / 150.5	66.4	[35.8, 123.5]
Overall	1,589	195	49	166 / 6400.0	25.9	[22.3, 30.2]
Any DSL (PTA > 25 dB HL, Visual Acuity > .3 logMAR)						
<65	358	34	9	4 / 1695.7	2.4	[0.9, 6.3]
65-74	697	63	10	42 / 3160.1	13.3	[9.8, 18]
75-84	430	82	21	58 / 1495.9	38.8	[30.0, 50.2]
85+	77	23	8	17 / 144.1	118.0	[73.3, 189.8]
Overall	1,562	202	48	121 / 6495.9	18.6	[15.6, 22.3]
HL and PCI (PTA > 25 dB HL, MMSE < 24)						
<65	354	34	8	5 / 1458.5	3.4	[1.4, 8.2]
65-74	757	65	9	12 / 2146.6	5.6	[3.2, 9.8]
75-84	535	85	29	6 / 798.2	7.5	[3.4, 16.7]
85+	130	32	13	1 / 78.2	12.8	[1.8, 90.7]
Overall	1,776	216	59	24 / 4481.5	5.4	[3.6, 8.0]

4.4 Discussion

4.4.1 Key Findings

The aims of this chapter were to report prevalence and incidence of Hearing Loss, Dual Sensory Loss and Hearing Loss co-morbid with cognitive impairment in Australia during the 1990s. Hearing Loss was highly prevalent among older adults. Notably, nearly nine out of ten adults aged older than 85 had some Hearing Loss, and half had moderate to severe levels of Hearing Loss. There were also no sex differences in prevalence among this older cohort. If these prevalence rates remained unchanged, based on current estimates of the age and sex distribution of the Australian population there would be 183,722 men and 261,967 women over the age of 55 with Dual Sensory Loss, and 91,342 men and 122,611 women with co-occurring hearing and cognitive impairment.

Previous analyses of BMES found an overall prevalence of 33% (hearing loss defined by PTA > 25 dB HL) for the entire sample (Gopinath, Rochtchina, et al., 2009). The authors did not report prevalence for narrow age-bands, though did observe that adults aged older than 80 years were 50 times more likely to have any degree of hearing loss, and 148 times more likely to have moderate hearing loss when compare to adults in their 50s. One other Australian study to report prevalence of hearing impairment also placed prevalence of better ear PTA > 25 dB HL to at 63% in adults aged 71 years and older (Wilson et al., 1999). These estimates are considerably lower than those calculated from the National Health and Nutrition Examination Survey (NHANES, USA), although this study only had data available for adults aged 20 to 69 years (Agrawal, Platz, & Niparko, 2008). The estimates reported here are comparable to, though slightly higher than more recent US estimates derived from NHANES data which placed prevalence in adults aged 70 years and older at 63.1% for $PTA_{0.5,1,2\,7\,5\,kHz}$ > 25 dB HL, and 26.5% for in the better ear $PTA_{0.5,1,2\,7\,5\,kHz}$ > 40dB HL in the better ear (Lin,

Thorpe, et al., 2011). Unlike Lin et al (2011), a sharp jump in prevalence between the 70-74 and 75-79 age groups was not found. The slightly higher prevalence reported here could be due to the inclusion of adults residing in institutions in both ALSA and BMES, and because racial groups with lower levels of hearing loss were included in NHANES. Other studies comparing age and cohort differences have reported that the prevalence of hearing loss was lower in younger generations (Zhan et al., 2009).

Co-occurrence of hearing loss with either vision loss or cognitive impairment was uncommon in adults younger than 75 years of age and did not differ between men and women, but co-morbid prevalence estimates were substantial for the oldest-old cohorts (aged 80 years and older). Incidence rates for co-morbid conditions were low, increased with age and did not differ between the men and women. Dual Sensory Loss prevalence based on self-report has been estimated to be 7.3% for adults aged 65 to 79, and 16.6% for adults aged 80 years and older (Caban et al., 2005), which are lower than the prevalence rates provided here. The prevalence of Dual Sensory Loss reported here is also greater than other estimates derived from clinical data. Smith and colleagues (2008), identified Dual Sensory Loss in 9% of adults aged between 75 and 84 years, and in 22% of adults aged 85 years and older. These differences can be attributed to differing definitions of Dual Sensory Loss and sample frame. Smith used a more conservative cut-point for hearing loss (PTA > 40 dB HL) and recruited veteran patients from a medical centre in Tennessee, USA. The more conservative cut-point was justified on the grounds that people with a moderate "degree of hearing impairment would likely have considerable listening difficulties in most situations without the use of amplification devices" (Smith et al., 2008, p. 601). On the other hand, it is possible that milder levels of hearing loss will have greater impacts on adults with vision loss. There is a need to develop an internationally agreed standard definition of Dual Sensory Loss.

4.4.2 Comparison with Self-Report Data

A second aim of this chapter was to compare findings in relation to audiometric hearing loss with self-rated hearing difficulty. Self-report data did not provide a reliable basis for estimating prevalence in the general population. In particular, self-reported hearing appeared to overestimate hearing impairment (PTA > 25 dB HL) in younger age cohorts while underestimating hearing impairment in older age cohorts. Although previous comparisons of self-report with audiometric measures have supported conclusions that self-report data may be sufficient for estimating overall prevalence of hearing loss, these studies only compared prevalence in broad age cohorts and failed to consider an age bias in self-reported health measures (Nondahl et al., 1998). Indeed, in this study the difference between self-report and audiometric based prevalence for all adults aged 55 years and older was minimal. Other studies have made similar conclusions that self-report data should not be used in place of audiometric measures where possible (Hong et al., 2011).

Social comparison theory (Willis, 1981) provides one explanation for the failure of self-reported hearing to detect age differences. Social comparison theory maintains that older adults tend to overrate their perceived health because they make implicit downward comparisons with negative old age stereotypes (Heckhausen & Brim, 1997; Sargent-Cox et al., 2008). The age bias inherent in self-reported hearing items could therefore reflect the downward social comparisons older adults are surmised to make when rating their health despite loss of functioning (Heckhausen & Brim, 1997). A similar explanation was given for the poorer performance of the HHIE in estimating prevalence in adults aged 65 years and older compared to adults aged between 48 and 64. Nondahl and colleagues (1998) suggested that older adults are more likely to be accepting of hearing impairment as they do not consider it an unusual aspect of ageing. The high prevalence and common experience leads to hearing loss becoming

normalised in older adults. Further, as hearing decline is generally a gradual process; many adults have time to adjust to hearing loss.

Younger age groups could over-estimate their hearing difficulties for a number of reasons. Firstly, they are more likely to be actively participating in the workforce and have greater work-related demands on their hearing. Certainly after the retirement age of 65 self report data no longer over estimates audiometric hearing impairment. Also, hearing ability for pure-tone-thresholds below 4kHz begin to decline in the 50s, and initial losses may be more noticeable at these ages (Wiley, Chappell, Carmichael, Nondahl, & Cruickshanks, 2008). Low levels of hearing aid utilisation in younger age groups could also contribute to the differences between self report and audiometric measures. It has been reported that adults may experience hearing difficulties for up to 10 years before they recognise their hearing to be a problem and access hearing services during their mid-70s (Davis et al., 2007). Finally, this could reflect a cohort effect whereby younger cohorts are more likely to report health problems and functional difficulties. Nor should it be discounted that the apparent over reporting to hearing loss is a real effect reflecting the poorer health status of younger cohorts. Seeman, Merkin, Crimmins & Karlamangla (2010) found significant increases in disability over a 16 year period in a cohort aged 60-69 years while those aged 70-79 years showed no significant changes in disability and those aged 80 years and over showed lower prevalence of functional limitations.

This is not to say that self-reports of sensory loss are not without value. The clinical measures used in this study were restricted to visual acuity and audiometric hearing thresholds. In contrast, perceived sensory loss may be sensitive to multiple components of visual and auditory functioning, such as stereo acuity, contrast sensitivity, and peripheral vision. Moreover, self-report may better reflect disability level whereas clinical measures tap into impairment level. It is therefore important to

recognise that self-report and objective measures are not identical or proxies for each other, but may be brought to bear on different though complementary research questions.

4.4.3 Limitations

There are some limitations with this data. The prevalence reported here are not representative of the current Australian adult population, as they are based on samples drawn during the early 1990s from Adelaide in South Australia, and the late 1990's from Blue Mountains in New South Wales. Small numbers in some cells may have resulted in unreliable incident rate estimates as reflected by wide confidence intervals, for example there were only 26 women aged 85 years or older with co-occurring cognitive impairment and hearing loss. The wide time intervals also mean the incidence rates are imprecise as the exact time of sensory loss onsets are unknown. Data on eye disease and other aspects of visual and auditory functioning were unavailable in the pooled data set. It has been suggested that less commonly assessed aspects of vision and hearing, such as contrast sensitivity and central auditory function are stronger predictors of independence and levels of sensory-related disability or functional limitations (Schneider & Pichora-Fuller, 2000). Finally, this chapter has focused on cognitive and sensory loss as impairment states, and not as disability or handicap states. It could strongly be argued that analyses pitched at the disability and handicap levels will have a greater contribution to our understanding of their psychosocial impacts on the health and well-being of older adults. Nevertheless, this descriptive study provides the most comprehensive population estimates of sensory loss amongst older Australians in the 1990s.

CHAPTER 5: Life Expectancies With and Without Sensory Impairment

Synopsis

Sensory impairments contribute significantly to non-fatal burden of disease at the population level, but no studies have used longitudinal data to quantify the number of years older adults may be expected to live with sensory impairment. This chapter reports on the use of interpolated Markov chain models to estimate sensory impaired life expectancies and transition rates between three health states: no impairment, sensory impairment, and death. Although men are often reported to have higher prevalence and incidence of sensory loss, the results indicated that on average, women actually experienced a greater number of years with vision impairment, hearing impairment and dual sensory impairment. At age 65, both men and women could be expected to live at least half their remaining years of life with at least a mild degree of Hearing Loss. However, at the same age men could expect to live 15% of their remaining years of life with impaired visual acuity whereas women could expect to live 20% of their remaining years of life with impaired visual acuity. Self-report measures were again shown to be inappropriate for deriving population level estimates of sensory impairment among older adult cohorts.

5.1 Background

The previous chapter reported population estimates of prevalence and incidence of sensory impairment derived from the pooled ALSA and BMES samples. The present chapter extends these findings by incorporating mortality data multi-state analyses that produce expected years lived with sensory loss in addition to prevalence and transition rates. Summary indices that combine mortality and morbidity information to express the expected years of life lived in disease or disability free states are commonly used to gauge the health of a population (McKenna, Michaud, Murray, & Marks, 2005) and are needed to evaluate compression of morbidity (Imai & Sone, 2007; Molla & Madans, 2008), making them useful for informing decisions on health spending and guiding health policy. Health-adjusted life expectancies (HALE) have classically been calculated using the Sullivan method (Sullivan, 1971), which combines prevalence data with life table mortality data. The Sullivan method is also used to estimate burden of disease as defined by Disability Adjusted Life Years (DALYs). These describe life lost due to premature death and life lost due to disability [DALY = Years of Life Lost(YLL) + Years Lived with Disability(YLD)] (Murray & Lopez, 1996, 1997). In the year 2000, adult onset hearing loss was the second leading cause of YLD globally (Mathers, Smith, & Concha, 2000).

5.2 Methods

5.2.1 Participants and Measures

This chapter uses the same pooled sample as in the previous chapter for the outcome measures of hearing loss, dual-sensory loss and self-reported hearing difficulty. However, estimates for vision impairment were based on a larger sample that included PATH, SOPS, ALSA, BMES and CLS.

5.2.2 Statistical Analyses

Sensory life expectancies were calculated with Interpolated Markov Chain software (IMaCh version 0.98k) (Jagger, Goyder, Clarke, Brouard, & Arthur, 2003; Lievre, Brouard, & Heathcote, 2003). IMaCh software estimates the average number of years lived with sensory impairment using a multi-state Markov model for discrete data to model transitions between three health states. In the context of this chapter, the three health states are: no sensory impairment, sensory impairment and death. There are four possible transitions between these health states, though the expected recovery from sensory loss is close to zero. Standard procedures for IMaCh analyses were followed. Observed time intervals were partitioned into smaller 1-month intervals to approximate an underlying continuous time process. Transition probabilities between the three health states were estimated by multinomial logistic regression conditioned on age and sex. The transition probabilities were then used as inputs to a multistate life-table. These models produced the expected number of years lived with sensory loss, the average proportion of life lived with sensory loss, observed and period stable prevalence estimates and annual transition probabilities for men and women for all ages between 55 to 95 years.

5.3 Results

Unfortunately IMaCh models for moderate Hearing Loss (PTA > 40 dB HL) did not produce reliable estimates. Prevalence estimates summed to values greater than 100%. Life expectancies were also unrealistic, exceeding 90 years for men aged 65 or younger, and 93 years for women aged 65 or younger. It is most likely this occurred due to small numbers of people with moderate hearing loss, particularly among younger ages. For this reason only results for hearing loss defined by PTA > 25 dB HL are reported.

5.3.1 Observed and Period Prevalence

The observed prevalence and period stable prevalence estimates are generated from the IMaCh model are presented in Figures 5.2-5.5. The period stable prevalence takes into account incidence and mortality rates, and reflects the proportion of the population expected to have sensory impairment on the first of January in the year 2000. The observed prevalence is the cross-sectional prevalence at baseline, and therefore is interpreted in a similar way to the prevalence estimates provided in Chapter 4. The period stable prevalence of hearing impairment, vision impairment and dual sensory loss was marginally less than the observed prevalence. In particular, it appears that the levels of visual impairment will decline slightly over time as the observed baseline prevalence was also above the 95% confidence interval for the period stable prevalence on January 1 2000. In contrast the observed prevalence for self-report hearing was lower than the period stable prevalence. The proportion of the population identified with clinically defined sensory impairment increased with age at comparable rates for men and women. The high prevalence of mild hearing impairment was again clearly evident for the oldest-old cohort. Note that the prevalence estimates provided in Chapter 4 were reported for 5-year age brackets up to the age of 84, and grouped all adults aged 85

years and older together. This is in contrast to the prevalence estimated by IMaCh which are given for each year of age.

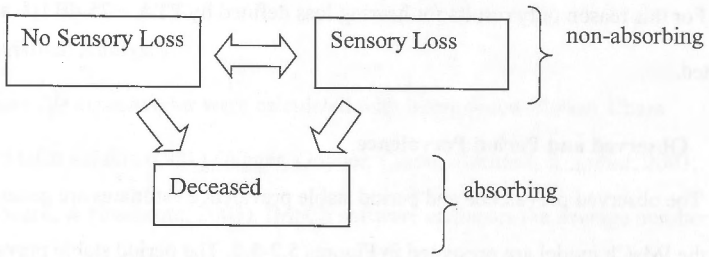


Figure 5.1 Three health states and four possible transitions between 2 non-absorbing states (no sensory loss and sensory loss) and 1 absorbing state (death). igure based on Lievre, Brouard, & Heathcote (2003)

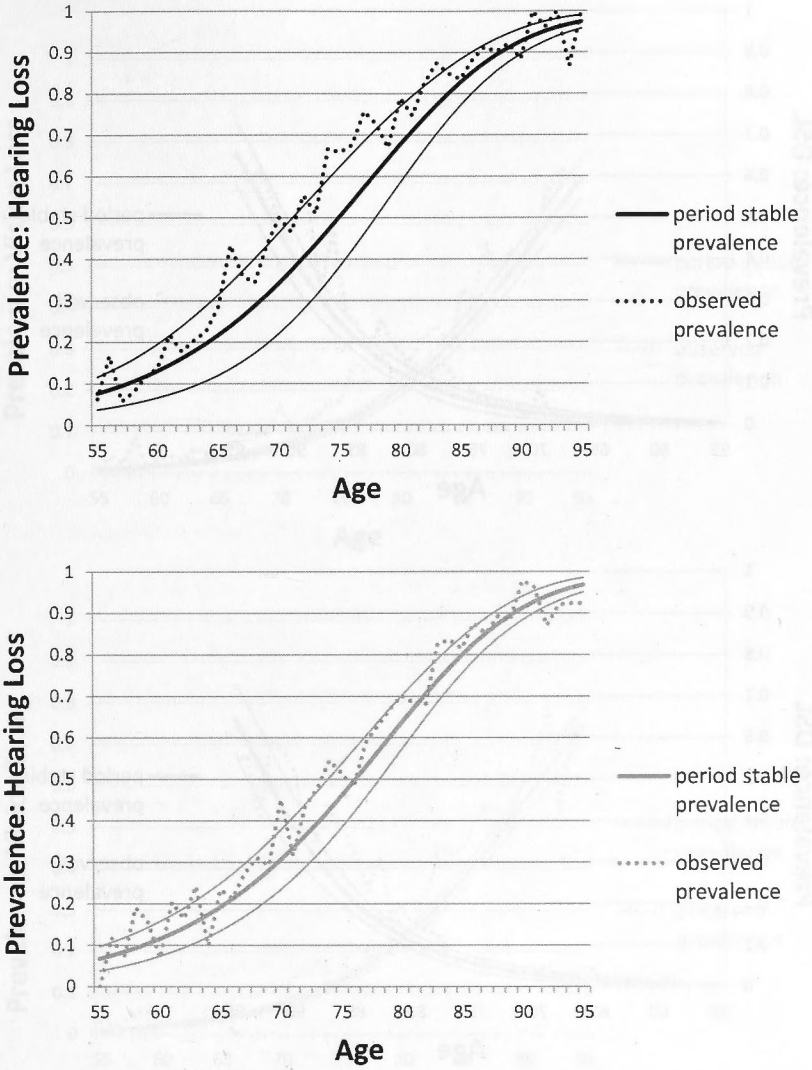


Figure 5.2 Observed (at baseline) and period stable (at 1/1/2000) prevalence of Hearing Loss (PTA > 25 dB) with 95% confidence intervals for men (black lines, top panel) and women (grey lines, bottom, panel).

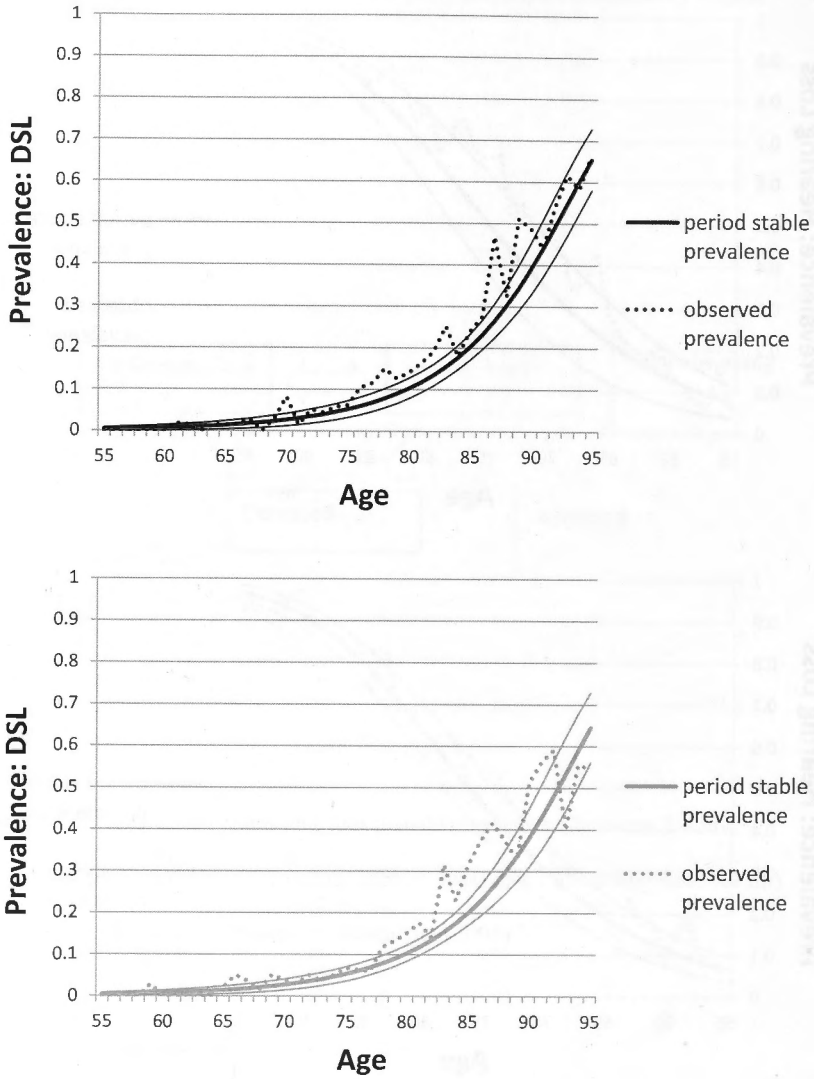


Figure 5.3 Observed (at baseline) and period stable (at 1/1/2000) prevalence of Dual Sensory Loss with 95% confidence intervals for men (black lines, top panel) and women (grey lines, bottom, panel).

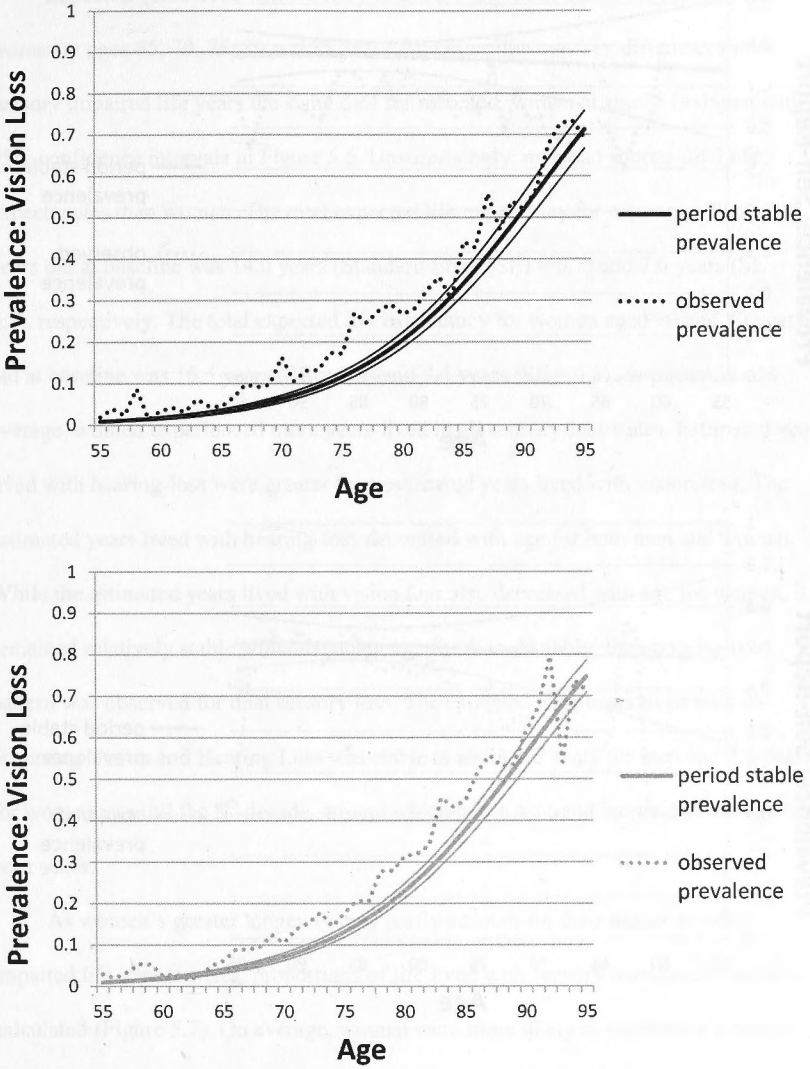


Figure 5.4 Observed (at baseline) and period stable (at 1/1/2000) prevalence of Vision Loss (> 0.3 logMAR) with 95% confidence intervals for men (black lines, top panel) and women (grey lines, bottom, panel).

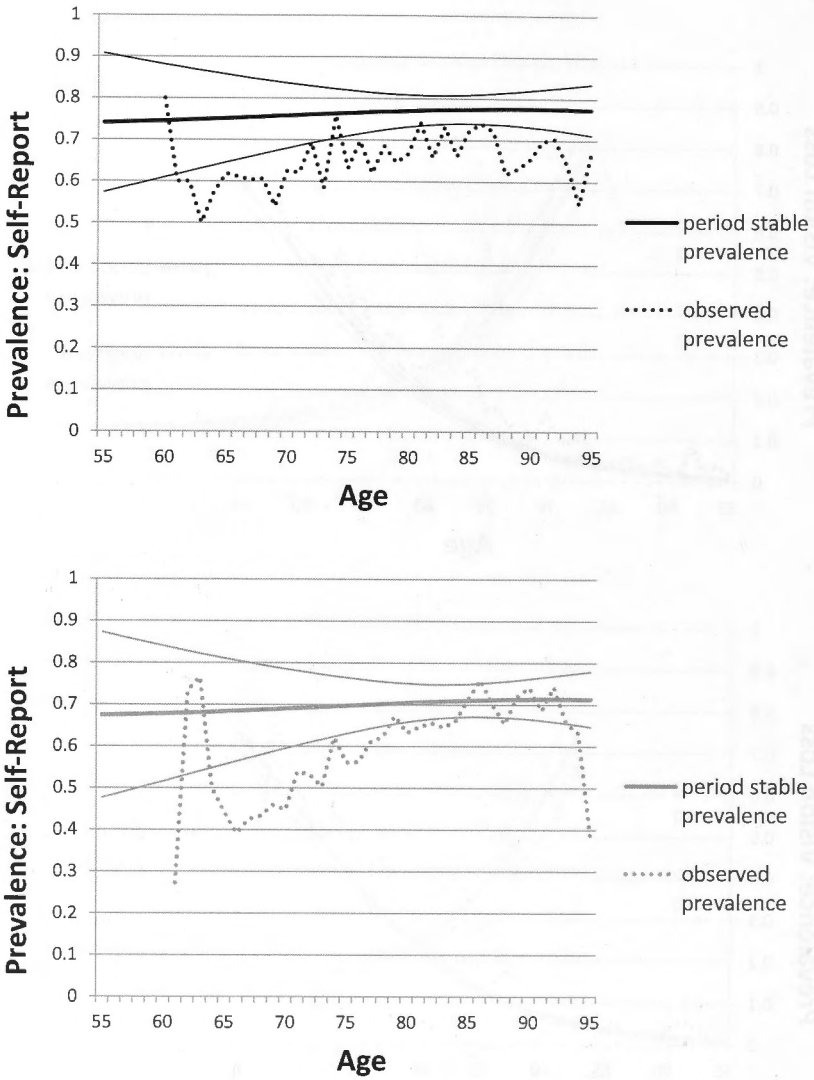


Figure 5.5 Observed (at baseline) and period stable (at 1/1/2000) prevalence of Self-Reported Hearing Difficulty with 95% confidence intervals for men (black lines, top panel) and women (grey lines, bottom, panel).

5.3.2 Sensory Life Expectancies

Expected years lived with sensory losses are shown in **Table 5.1** for men and women at ages 65, 70, 75, 80 and 85 years. To better illustrate sex differences in sensory impaired life years the same data for men and women at age 75 is shown with 95% confidence intervals in Figure 5.6. Unsurprisingly, men had shorter total life expectancies than women. The total expected life expectancy for men aged 70 and 80 years old at baseline was 14.0 years (Standard Error (SE) = 0.7) and 7.6 years (SE = 0.3), respectively. The total expected life expectancy for women aged 70 and 80 years old at baseline was 16.5 years (SE = 0.4) and 9.4 years (SE = 0.3), respectively. On average, women experienced more years lived in all sensory-loss states. Estimated years lived with hearing-loss were greater than estimated years lived with vision-loss. The estimated years lived with hearing-loss decreased with age for both men and women. While the estimated years lived with vision loss also decreased with age for women, it remained relatively stable with advancing age for men. Notably, the opposite age pattern was observed for dual sensory loss. The expected total years lived with co-occurring Vision and Hearing Loss was stable at about 1.5 years for men and 2.6 years for women up until the 8th decade, around which point the trend increased with age for both sexes.

As women's greater longevity may partly account for their longer sensory impaired life expectancies, proportions of life lived with sensory impairment were also calculated (Figure 5.7). On average, women were more likely to experience a greater proportion of life lived with vision loss and dual sensory loss. However, there did not appear to be any sex differences in the estimated proportional life expectancies for audiometric Hearing Loss, and men had greater estimated proportional life expectancies for self-reported hearing

Table 5.1 Sensory impaired life expectancies (years) for men and women aged 70, 75, 80, and 85 years old.

		Total Life Expectancy*		Years lived with Vision Loss		Years lived with Hearing Loss		Years lived with Hearing Difficulty		Years lived with Dual Sensory Loss	
Sex	**Age**	**Years**	**(SE)**	**Years**	**(SE)**	**Years**	**(SE)**	**Years**	**(SE)**	**Years**	**(SE)**
Men											
	65	17.7	(0.7)	2.5	(0.1)	8.9	(0.8)	10.6	(0.5)	1.5	(0.2)
	70	14.0	(0.7)	2.5	(0.1)	8.3	(0.7)	8.3	(0.3)	1.6	(0.1)
	75	10.6	(0.5)	2.5	(0.1)	7.4	(0.5)	6.3	(0.2)	1.6	(0.1)
	80	7.6	(0.3)	2.5	(0.1)	6.1	(0.3)	4.7	(0.1)	1.7	(0.1)
	85	5.2	(0.2)	2.4	(0.1)	4.7	(0.2)	3.4	(0.1)	1.8	(0.2)
Women											
	65	20.6	(0.4)	4.0	(0.2)	10.7	(0.5)	11.4	(0.7)	2.7	(0.2)
	70	16.5	(0.4)	4.0	(0.2)	10.0	(0.5)	9.1	(0.4)	2.7	(0.2)
	75	12.7	(0.4)	3.9	(0.2)	9.0	(0.4)	7.0	(0.3)	2.7	(0.3)
	80	9.4	(0.3)	3.7	(0.2)	7.6	(0.3)	5.3	(0.2)	2.8	(0.3)
	85	6.6	(0.2)	3.3	(0.1)	5.9	(0.2)	3.9	(0.2)	3.1	(0.4)

Vision Loss: Visual acuity > 0.3 logMAR

Hearing Loss: PTA > 25 dB HL

Dual Sensory Loss: PTA > 25 dB, and Visual acuity > 0.3 logMAR

Hearing Difficulty: Self-report of hearing difficulties

***Note:** Total life expectancies estimated from Hearing Loss only model.

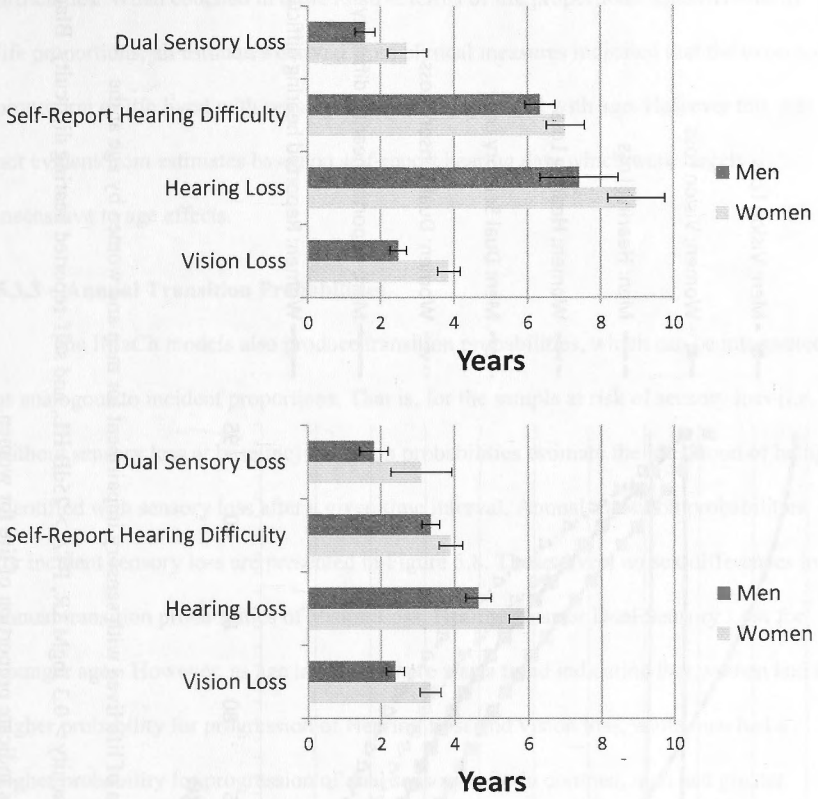


Figure 5.6 Expected years lived with sensory impairment for men and women at age 75 (top panel) and age 85 (bottom panel) with 95% confidence intervals.

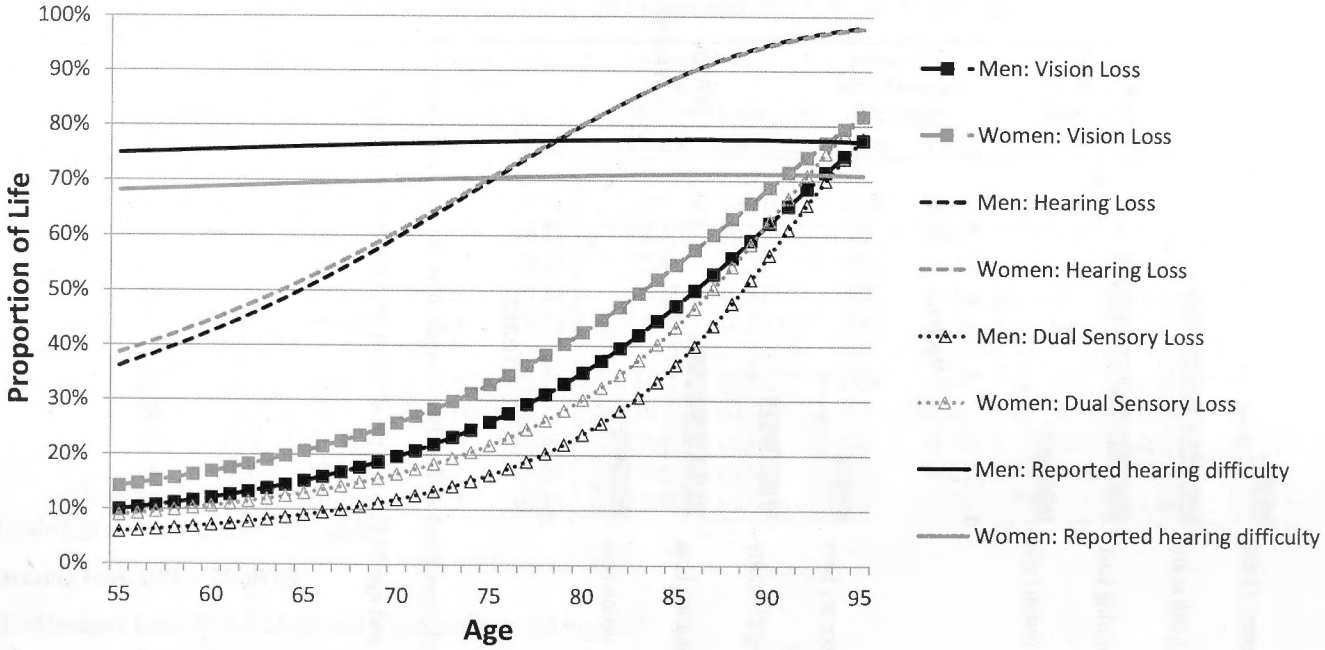


Figure 5.7: IMaCh estimates of average proportion of life lived with sensory impairment for men and women by age at the population level. Sensory impairments are visual acuity > 0.3 logMAR, PTA > 25dB HL, and self-reported hearing difficulty. Black lines indicate proportion of life for men; grey lines indicate proportion of life for women.

difficulties. When couched in these relative terms of life proportions relative terms of life proportions, all estimates derived from clinical measures indicated that the expected proportion of life lived with sensory impairment increased with age. However this was not evident from estimates based on self-report hearing data which were largely insensitive to age effects.

5.3.3 Annual Transition Probabilities

The IMaCh models also produce transition probabilities, which can be interpreted as analogous to incident proportions. That is, for the sample at risk of sensory loss (i.e. without sensory loss at baseline) transition probabilities estimate the likelihood of being identified with sensory loss after a given time interval. Annual transition probabilities for incident sensory loss are presented in Figure 5.8. These reveal no sex differences in annual transition probabilities of Vision Loss, Hearing Loss or Dual Sensory Loss for younger ages. However, as age increased there was a trend indicating that women had a higher probability for progression of Hearing Loss and vision loss, while men had a higher probability for progression of dual sensory loss. In contrast, men had greater transition probabilities for self-reported hearing difficulty at all ages. These differences were not tested for statistical significance by IMaCh. Again, the estimates derived from self-report measures were less sensitive to age effects relative to clinical measures.

The probability of transitioning to dual sensory loss over a 1-year period did not exceed 0.01 until age 67 for men, and age 69 for women. As a way of validating the method used to estimate incidence ratios, it is useful to compare the transition probability of dual sensory loss with the incident rates reported in the previous chapter. The probability of converting to dual sensory loss after one year was 0.09 for men aged 85 years old and 0.07 for women aged 85 years old. These estimates are consistent with the lower bound of the 95% confidence interval for the incident rates of dual sensory loss, which were 132.6 per 1000 person-years (95% CI: 85.6, 205.6) for men and 118.0

per 1000 person-years (95% CI: 73.3, 189.8) for women aged 85 years and older. The upper bands of the confidence intervals also align with the annual transition probabilities for men (0.21) aged 95 years old, and women (0.19) aged 95 years old. It should be noted when interpreting these estimates that incident rates and transition probabilities are not equivalent.

The annual transition probabilities for all possible transitions between health states are given in Figures 5.9-5.12. The probability of transitioning from an impaired sensory state to an unimpaired sensory state (remission) declined with age, and in the case of Hearing Loss was close to zero for all ages. This contrasted with the probability of reporting no hearing difficulty one year after previously reporting a hearing difficulty, which whilst also low at around 5.7% for men and 6.7% for women at age 80, appeared to be relatively stable with increasing age.

The mortality probability increased with age in the single sensory loss models. However, the mortality probability for adults with dual sensory loss was constant at around 20% for all ages, with a slight increase for men after the age of 80. Interestingly, the pattern of mortality probabilities for clinically defined sensory loss differed to mortality probabilities for self-report hearing difficulty. Whereas the probability of death increased with age at a faster rate among adults identified with Vision Loss or Hearing Loss relative to adults with no sensory loss, there was no discernible difference in the probability of death at any age between adults reporting hearing difficulties over background noise and adults who did not report such hearing difficulties. This indicates that self-report measures of sensory loss may be less sensitive to mortality risk than objective measures of actual sensory function.

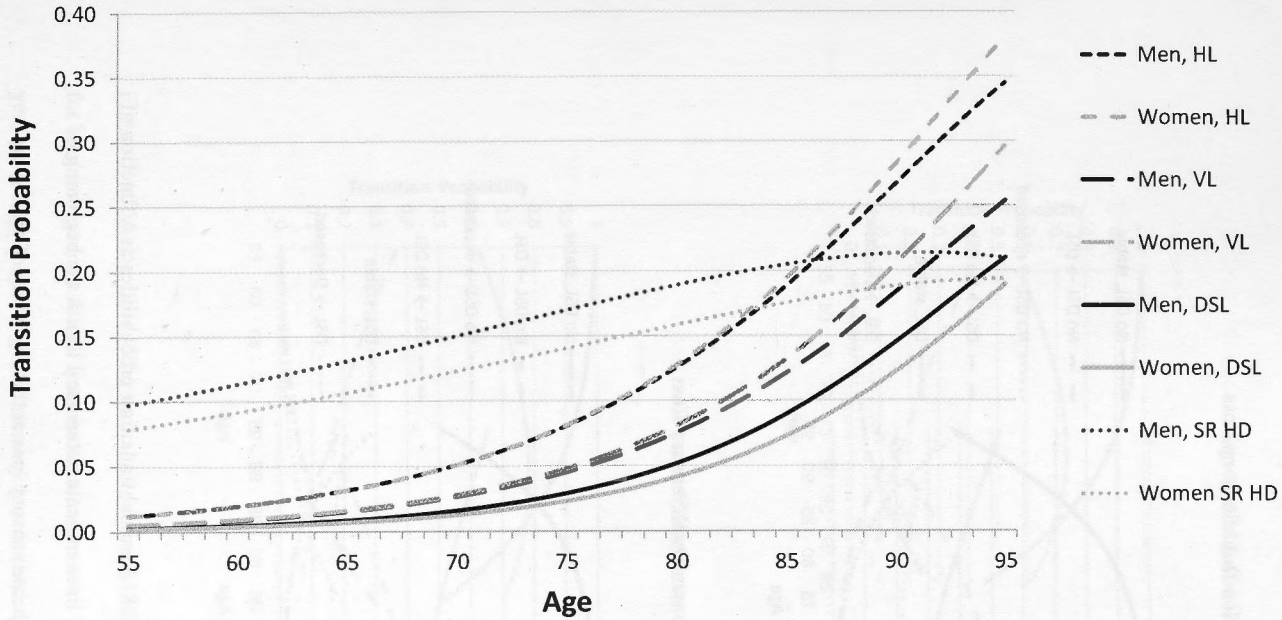


Figure 5.8 Annual transition probabilities as a function of age for conversion from 'No Impairment' to either 'Hearing Loss' (HL), 'Vision Loss' (VL), 'Dual Sensory Loss' (DSL), or 'Self-Reported Hearing Difficulty' (SR HD). Black lines indicate probability of transition for men; grey lines indicate probability of transition for women.

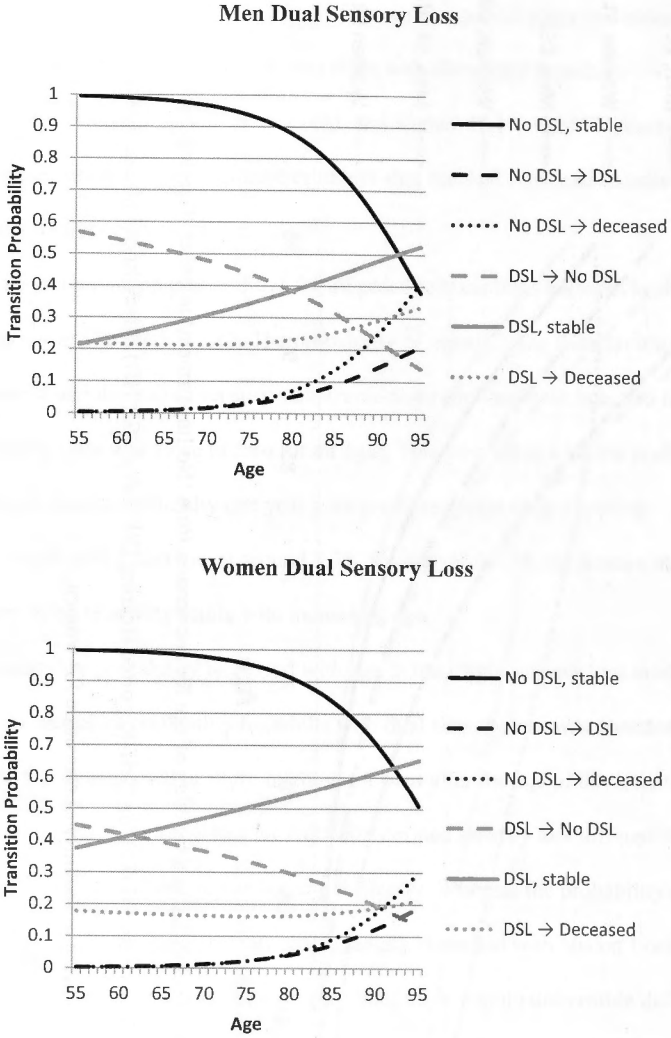
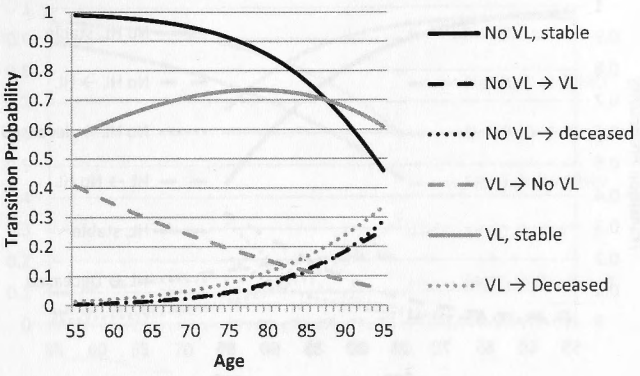


Figure 5.9 Dual Sensory Loss (DSL) annual transition probabilities as a function of age, for men and women. Black lines indicate no sensory loss at the beginning of the 1-year period; grey lines indicate sensory loss at the beginning of the 1-year period. Solid lines show probability of no transition, dashed lines show probability of transition, dotted lines show probability of death.

Men Vision Loss



Women Vision Loss

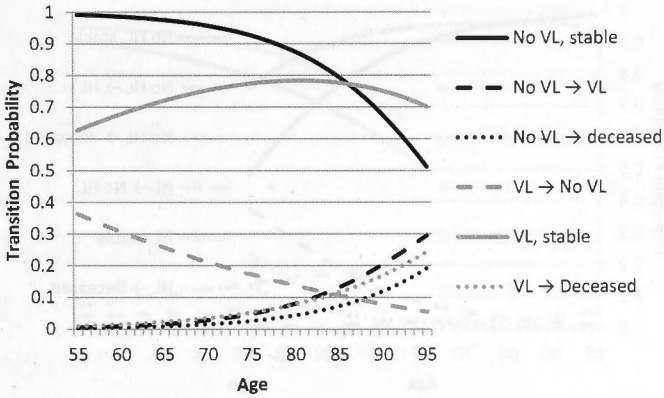
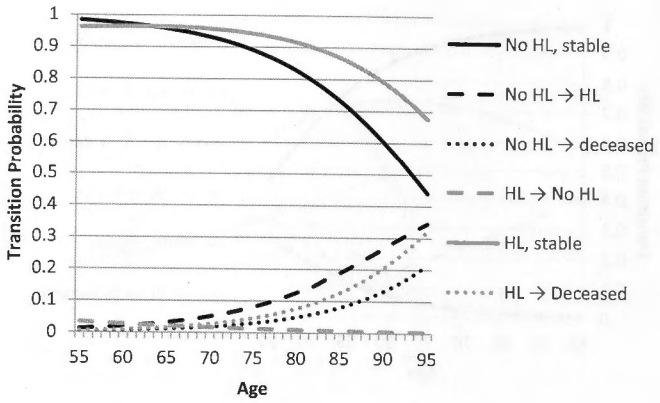


Figure 5.10 Vision Loss (VL) annual transition probabilities as a function of age, for men and women. Black lines indicate no sensory loss at the beginning of the 1-year period; grey lines indicate sensory loss at the beginning of the 1-year period. Solid lines show probability of no transition, dashed lines show probability of transition, and dotted lines show probability of death.

Men Hearing Loss



Women Hearing Loss

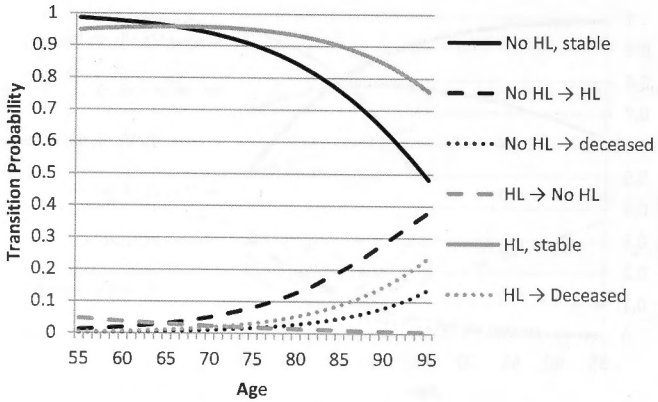


Figure 5.11 Hearing Loss (HL) annual transition probabilities as a function of age for men and women. Black lines indicate no sensory loss at the beginning of the 1-year period; grey lines indicate sensory loss at the beginning of the 1-year period. Solid lines show probability of no transition, dashed lines show probability of transition, and dotted lines show probability of death.

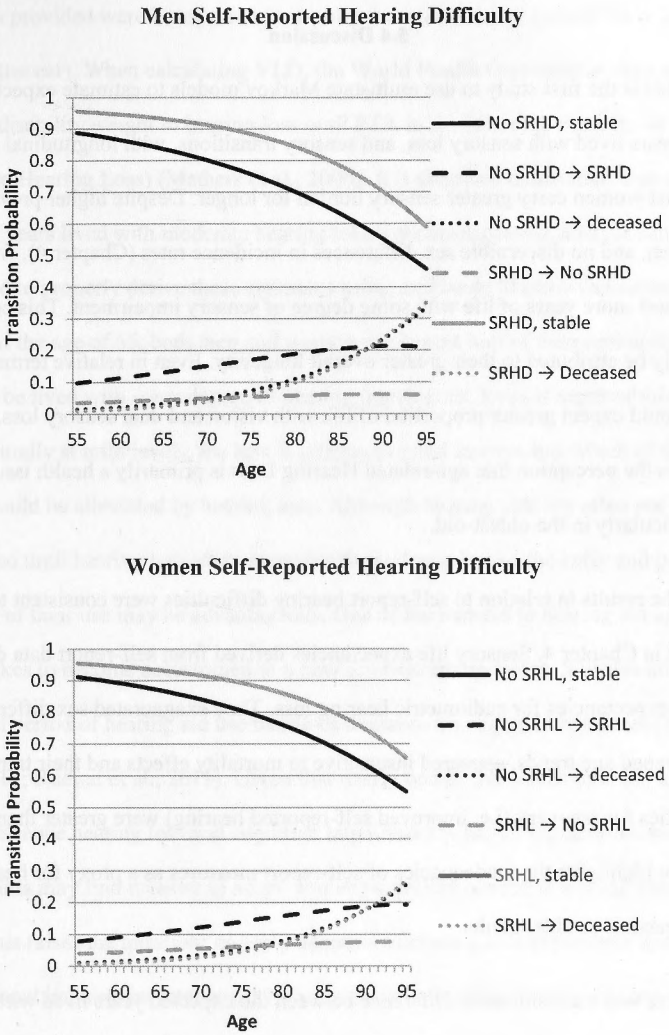


Figure 5.12 Self-Reported Hearing Difficulty (SRHL) annual transition probabilities as a function of age for men and women. Black lines indicate no sensory loss at the beginning of the 1-year period; grey lines indicate sensory loss at the beginning of the 1-year period. Solid lines show probability of no transition, dashed lines show probability of transition, and dotted lines show probability of death.

5.4 Discussion

This is the first study to use multistate Markov models to estimate expected number years lived with sensory loss, and sensory transitions, with longitudinal data. It is clear that women carry greater sensory burden for longer. Despite higher prevalence among men, and no discernible sex differences in incidence rates (Chapter 4), women could expect more years of life with some degree of sensory impairment. This could most likely be attributed to their greater overall longevity. Even in relative terms, women could expect greater proportion of life with vision and dual sensory loss. This challenges the perception that age-related Hearing Loss is primarily a health issue for men, particularly in the oldest-old.

The results in relation to self-report hearing difficulties were consistent to those presented in Chapter 4. Sensory life expectancies derived from self-report data diverged from life expectancies for audiometric hearing loss. They exaggerated sex differences, had dampened age trends, appeared insensitive to mortality effects and their transition probabilities for recovery (i.e. improved self-reported hearing) were greater than zero. This again highlights the inadequacies of self-report measures as a proxy for hearing acuity measures in older adults.

There was a considerable difference between the expected years lived with vision loss compared to the years lived with hearing loss. As vision and hearing comprise two distinct sensory modalities it is not unreasonable to expect such differences, however there are two additional reasons underlying this discrepancy that are worth mentioning. Firstly, visual acuity was tested under optimal conditions and corrected by prescription glasses or contact lenses. This is in contrast to hearing thresholds which were not corrected by use of hearing aids. Secondly, the cut-point used to define hearing impairment could be conservative relative to the definition of vision impairment. The

estimates provided were based on the cut-point for *mild* Hearing Loss (PTA > 25dB HL in the better ear). When calculating YLD, the World Health Organisation does not assign a disability weight to hearing loss until PTA in the better ear exceeds 40 dB HL (moderate Hearing Loss) (Mathers et al., 2000). It is therefore unfortunate that attempts to model years lived with moderate hearing loss were unsuccessful, a larger sample may be needed to properly derive these estimates using multistate Markov techniques.

At the age of 65, both men and women can expect half of their remaining years of life to be lived with some degree of hearing impairment. Even if much of this hearing loss is initially at mild levels, the loss is progressive and irreversible. Much of the future burden could be alleviated by hearing aids. Although hearing aids are often not considered until hearing loss starts to approach moderate levels, the early and persistent adoption of their use may be advantageous. One of the barriers to hearing aid use is the time it takes to become accustomed to a new acoustic environment. It is also known that the initial period of hearing aid use increases demands on cognitive resources (Fischer et al., 2011; Rudner et al., 2011). Given that nearly one in five adults over the age of 85 may experience hearing loss and cognitive impairment (Chapter 4), early adopters of hearing aids may find it easier to adapt, and show greater benefit in hearing aids in later years. This raises the question; do early adopters of hearing aids experience lower levels of functional impairment later in life? The association between hearing aid use and rates of cognitive decline will be explored in a subsequent chapter of this thesis.

There have been few studies that have applied multi-state models to investigate transitions rates of sensory impairment. One study used a similar meta-analytic approach to report on the progression of hearing loss for individual frequencies (Chao & Chen, 2009), to show that progression to impaired levels was faster for higher frequencies, men, the left ear (for younger ages) and older adults. Chao and Chen (2009) did not include mortality data, and so were unable to model life years, and their

reliance on published effect estimates also resulted in strict assumptions concerning time and the specification of transition paths. Chapter 7 of this thesis will examine similar research questions using liner mixed models. Jagger and colleagues (2007) have used IMaCh to estimate differentials for years lived with functional disability among adults and found that there was little impact of sensory loss. However, Jagger defined sensory losses on the basis of interviewer judgements that were unlikely to be informed by a set of validated criteria. Future research should investigate how clinical measures of sensory loss impact on disability free life expectancies, and also further explore differentials for sensory impaired life expectancies (e.g. diabetes, occupation, noise exposure, and lifestyle).

There has been debate on the appropriateness of disease burden indicators which aggregate morbidity and mortality data. Robine (1998) has questioned how a complex system, such as the health of a population, can be adequately expressed by a single index. Advocates of Sullivan method summary indicators maintain that they are useful for assessing the implications of increased longevity, and also guiding policy decisions (Hyder, Rotllant, & Morrow, 1998; Morrow, Hyder, Murray, & Lopez, 1998). However, this fails to address other concerns that DALYs are value laden, biased towards fatal disease and that making resource allocation decisions based on such indices can lead to inequitable outcomes (Anand & Hanson, 1997), IMaCh models address many of these concerns (Jagger et al., 2007). Unfortunately, the analyses presented in this Chapter do not explicitly make a direct link between sensory loss and difficulties with activities of daily living. Thus, this chapter is pitched at the level of impairment and not disability, so the impacts of sensory loss on activity limitations cannot be compared to other health conditions.

5.4.1 Limitations

There are a number of limitations to the findings presented in this chapter. These estimates apply at the population level, and will not necessarily apply at the level of the individual. For example, it is not the case that all 65 year old men will experience 8.9 years of mild hearing loss. It is also unclear if the transition rates and life expectancies can be generalized for future cohorts of older adults. Perhaps of greater concern are limitations regarding the methods used and temporal data structure. ALSA participants were tested in their homes by trained nurses but not audiologists or ophthalmologists. It has been argued that between-group differences in incidence rates derived from Markov chain transitions modelled over wide time intervals (typically 12 months apart of greater) are likely to be biased and should be interpreted with extreme caution (van den Hout & Matthews, 2010; Wolf & Gill, 2009). This is due to the possibility of short period transitions of either recovery or disability that occur between measurement waves and are therefore unobserved. There is a 6 year interval between wave 2 and 3 in ALSA where true states of sensory impairment are unknown and approximated in the model. A similarly long time interval occurred for BMES (However, it might be argued that such biases are minimal in this instance as recovery from sensory impairment is unlikely.) On the other hand, there have been suggestions that outcomes with low recovery may be more optimally estimated with recently developed and more flexible multi-state models (Jackson, 2011; Meira-Machado, de Uña-Álvarez, Cadarso-Suárez, & Andersen, 2009) that allow recovery transitions to be constrained to zero. These caveats notwithstanding, this is the first attempt to estimate years lived with sensory impairment using multi-state models of longitudinal data.

CHAPTER 6: Sex Differences in Mortality Risk of Hearing Loss, Dual Sensory Loss and Rates of Change in Hearing

Synopsis

The association between hearing loss and mortality risk is largely explained by health, physical functioning, cognition, socio-demographics and other factors. In contrast, visual impairment and dual sensory loss have each been identified as independent risk factors for mortality. However, there are methodological and theoretical aspects of hearing-survival relations that are yet to be empirically investigated. Though hearing thresholds are continuous variables, they are often dichotomized or polytimised to aid interpretability, which may be at the cost of reduced power and increased error variance. Further, despite sex differences in longevity and reported interactions between hearing loss and sex when predicting survival time, few studies have conducted survival analyses separately for men and women. Questions concerning links between rates of change in hearing and survival also remain to be answered. Though there is some evidence to suggest that hearing decline is associated with increased mortality, true longitudinal hearing trajectories have not been tested as independent predictors of hearing. This chapter reports original analyses of pooled ALSA and BMES data that extend our understanding of hearing-survival relations by 1) comparing sex differences, 2) modelling hearing thresholds as a linear predictor, and 3) incorporating estimates of level and change in hearing as covariates in survival models.

Three approaches to accounting for study effects were also compared, namely: meta-analyses, study stratified analyses and study adjusted analyses. Results indicated that neither hearing thresholds nor dual sensory loss were associated with mortality in men. However, hearing thresholds and dual sensory loss were independent predictors of mortality in women. Rate of change in hearing was not reliably associated with increased mortality risk after adjusting for other risk factors. There were negligible differences between the three approaches to addressing study effects.

6.1 Background

6.1.1 Hearing Loss and Mortality

There is little evidence of an independent direct link between hearing loss and mortality. Whilst many studies have consistently reported univariate associations, the effect of hearing on survival time has generally been found to be explained or mediated by age, cognition, and other chronic health conditions. For example, a study of adults aged between 55 and 75 did not identify either self-reported or audiometric hearing loss as independent predictors of 10-year mortality (Mui, Reuben, Damesyn, Greendale, & Moore, 1998, cited in Mathers, Smith, & Concha, 2001; Reuben, Mui, Damesyn, Moore, & Greendale, 1999). Ostbye and colleagues reported that significant univariate associations between hearing impairment and five-year mortality were greatly attenuated and non-significant after adjusting for potential confounds in an older cohort of adults aged 65 years and older (Ostbye, Steenhuis, Wolfson, Walton, & Hill, 1999). Although both self-reported and clinically assessed hearing were tested as predictors, it was not clear how hearing impairment was actually defined by Ostbye et al. (1999). Further, the authors did not conduct survival analyses; rather they modelled vital status (alive or deceased) as the dependent variable in logistic regression. Lower survival time among adults with post-lingual deafness has also been attributed to self-reported health (Barnett & Franks, 1999). On the basis of findings such as these, the World Health Organization model of disease burden (Mathers, Smith, & Concha, 2001) assumes no relative risk for mortality.

Two DYNOPTA studies with audiometric data have previously examined hearing-related mortality in older adults and found similar results to Ostbye et al (1999) and Barnett et al (1999). An investigation of potential pathways between age-related hearing loss and mortality in a structural equation modelling framework using data from

BMES revealed that there was a reliable association between level of hearing loss and mortality risk, but that this was fully mediated by walking limitations, cognitive impairment and self-rated health (Karpa et al., 2010). Finally, hearing thresholds were again found not to be independent risk factors for mortality in ALSA (Anstey, Luszcz, Giles, & Andrews, 2001). However, Anstey et al. (2001) did report that two-year decline in hearing thresholds was predictive of mortality after adjusting for health and socio-demographics. In this study, two-year change in change in hearing was defined as a binary variable of decliners and non-decliners from two waves of data.

Unlike hearing loss, vision loss and age-related eye disease, such as cataract, have been reported to be independent risk factors for increased mortality risk (Thompson, Gibson, & Jagger, 1989; Wang, Mitchell, Simpson, Cumming, & Smith, 2001.). Further, visual impairment has been observed to have both direct and indirect effects on mortality (Christ, Lee, Lam, Zheng, & Arheart, 2008). In regards to dual sensory loss, there have been few studies of how co-occurring hearing and vision impairment impact on mortality risk. Self-report dual sensory loss is independently associated with increased mortality in the US based National Health Interview Survey (NHIS) (Lam, Lee, Gomez-Marin, Zheng, & Caban, 2006; Lee et al., 2007). Interestingly, Lee et al (2007) and Lam et al (2006) conducted their analyses separately for men and women, and found stronger effects for women. However, they were unable to control for lifestyle behaviour and disease variables like smoking and diabetes. Lee et al. (2007) suggested that these factors may explain the link between dual sensory loss and mortality. It has been suggested that attempts to investigate links between dual sensory loss and mortality using clinical measures have been limited by low statistical power or short follow up periods (Laforge, Spector, & Sternberg, 1992).

Given women's greater longevity and better hearing compared to men, it might be expected that any link between sensory function and mortality may differ for men

and women. The pattern of age related hearing loss differs between men and women, with women generally found to have better high frequency hearing but poorer low frequency hearing with advancing age (Murphy & Gates, 1997; See also Chapter 7 of this thesis). With the exception of Ostbye et al. (1999), all other studies described above that focused on hearing loss included sex as a covariate in their analyses, but none included interaction terms between sex and hearing or conducted their analyses separately for men and women. Studies that have tested if sex modifies the association between sensory impairment and survival time in older adults, have not obtained consistent findings. Poor performance on a whispered voice test was independently predictive of 6 year mortality in men but not in women (Appollonio, Carabellese, Magni, Frattola, & Trabucchi, 1995). A recent study using pooled Australian data found no sex differences in the association between self-reported sensory difficulties and mortality (Lopez et al., 2011). It is notable that neither of these two studies operationalized hearing loss using clinical definitions, rather they analysed sensory measures that were claimed to be more functional and ecologically valid. The value of sub-group analyses has previously been demonstrated by Gambassi (1999) who focused on a clinical sample and found that, in contrast to null findings from large population based studies, hearing loss was associated with mortality among low severity dementia patients (Gambassi et al., 1999). To the authors knowledge, no study has rigorously investigated sex differences in the association between audiometric hearing loss and mortality.

Despite the variety of approaches to defining hearing loss, results in relation to mortality have been remarkably consistent. However, no studies have analysed hearing thresholds as a linear variable. Studies of objective hearing measures of pure-tone audiometry have used a pure tone average (PTA) of 0.5, 1, 2 and 4 kHz greater than 25 dB HL in the better ear (Karpa et al., 2010), or PTA of 1 and 2 kHz greater than 40dB in

one ear (Reuben et al., 1999). Anstey et al (2001) analysed quintiles of hearing thresholds averaged across both ears for frequencies of 2, 3 and 4 kHz. It is possible that formatting audiometric measures as binary or dummy coded ordinal variables could obscure a relation between age-related hearing loss and mortality.

No studies have investigated associations between long-term rates of decline in hearing and all-cause mortality by applying joint parameter longitudinal survival analyses to repeated measures data over an extended period. Incorporating growth curve random effects for intercept and slope as covariates in survival analyses has been used to investigate if individual trajectories of change in a range of cognitive domains are predictive of mortality (Batterham, Mackinnon, & Christensen, 2011; Ghisletta, 2008; Ghisletta & Lindenberger, 2000) and dementia diagnoses (McArdle, Small, Backman, & Fratiglioni, 2005). All of these studies have reported null findings that rate of change in cognition was not associated with increased risk with the outcome of interest. Joint parameter longitudinal-survival models have advantages over more traditional difference score approaches to modelling change-survival relations as they allow for the inclusion of participants who only participate in a single wave, do not confound level and change information, and test for between person differences in change (Ghisletta, 2008).

6.1.2 Study Aims

On balance, level of hearing loss does not appear to be an independent risk factor for mortality, though there is some evidence to suggest that decline in hearing may be. It is the contention of this chapter that this is an aspect of hearing epidemiology worth pursuing further as there remain some research questions regarding links between hearing and mortality that have not yet been explored. The purpose of this study is to address three such unresolved questions using clinically defined measures of hearing

and vision. These concern the possibility of sex differences in sensory-mortality associations, the practice of formatting hearing thresholds as categorical variables which mask any relation between hearing and mortality, and whether actual rate of change in hearing is a risk factor for mortality independent of initial hearing level. Finally, as this study draws on pooled and harmonised data, this chapter will also demonstrate and compare three different approaches to addressing study effects when analysing harmonised data.

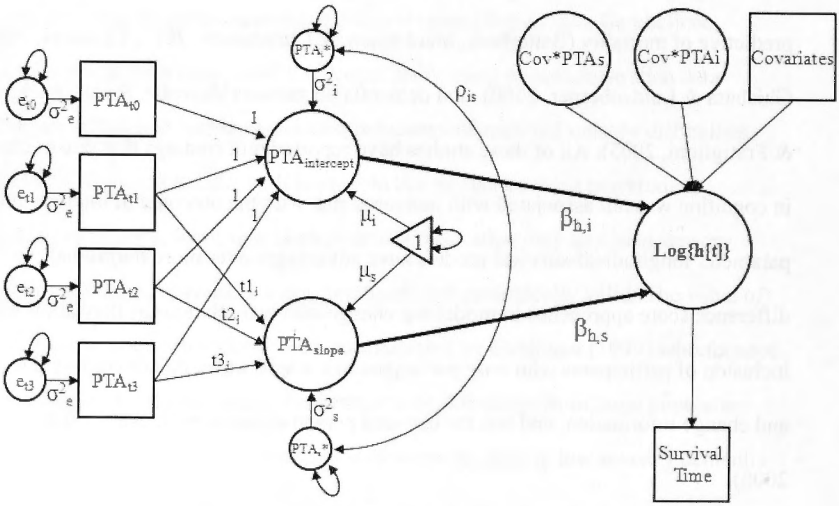


Figure 6.1 Schematic of joint parameter growth curve and survival model used to test for associations between rates of change in hearing with all-cause mortality risk. The PTA slope factor is estimated by individually varying time points.

6.2 Methods

6.2.1 Participants

This study used participants who completed hearing assessments from the ALSA waves 1, 3, 6 and 7 and BMES waves 2 and 3.

6.2.2 Variables

The dependent variable was survival time in years. Surviving participants were right censored if they were not known to be deceased at the time of the most recent linkage with the national death index. The primary independent variables were hearing thresholds, range of hearing loss and dual sensory loss. Hearing thresholds were defined by PTA in the better ear. Mild and moderate ranges of Hearing Loss were defined by PTA > 25 dB HL and PTA > 40 dB HL, respectively. Dual Sensory Loss was defined by standard ranges of functioning (Visual Loss > .3 logMAR, any hearing impairment by PTA > 25 dB HL). Other covariates included age, sex, study, education, MMSE < 24, corrected distance visual acuity, medical conditions (stroke, hypertension and diabetes), smoking status (current, former, never), self-rated health (excellent, good, fair, poor) and self-reported occupational noise exposure (5 years or more).

6.2.3 Analyses

To graphically illustrate differences in survival time by study and level of sensory loss, unadjusted as well as age and sex adjusted, Kaplan-Meier survival function curves were created for study (ALSA, BMES), Hearing Loss ('normal hearing', 'mild hearing loss' and 'moderate hearing loss') and for Dual Sensory Loss ("no sensory loss", "vision loss only", "hearing loss only", and "dual sensory loss"). The proportional hazards assumption was assessed statistically with chi square tests, and graphically by plotting scaled Schoenfeld residuals (Cleves, Gutierrez, Gould, & Marchenko, 2008) for hearing thresholds over survival time.

Cox proportional hazards models were run on the whole baseline sample and separately for men and women. Univariate, age and sex adjusted, and full multivariate adjusted models were tested, with the key independent variables of interest being one of either: 1) Hearing thresholds (linear), 2) Hearing Loss (categorical), or 3) Dual Sensory Loss (categorical). The full multivariate model included age, sex, smoking status, education, self-rated health, self-reported clinical diagnoses of diabetes, hypertension, history of stroke, MMSE < 24, and corrected distance visual acuity. As a final step, age, sex, study, visual acuity and MMSE interaction terms with hearing thresholds were also tested. All categorical variables were dummy coded. To aid interpretation of anticipated small hazard ratios, hearing thresholds (dB) were divided by 10, so that a hazard ratio of 1.05 would indicate a 5% increased mortality risk for a 10 dB increase in hearing thresholds. To address study effects models were either 1) fit directly to the pooled data and adjusted for study, 2) fit directly to the pooled data and stratified by study, and 3) conducted separately within each study and pooled estimates were calculated by standard meta-analysis techniques.

To test if longitudinal trajectories of PTA were predictive of all-cause mortality risk I simultaneously fitted latent growth curves with individually varying time points to 11-year PTA data, and incorporated the estimated intercept and slope factors as covariates in a Cox proportional hazards model within a structural equation modelling framework (Figure 6.1). Hazard ratios for the intercept and slope factors are interpreted as the risk of mortality associated with an increase of 1 dB from the sample mean intercept and sample mean slope, respectively. A series of analyses were conducted: an unadjusted univariate analysis (model 1), a multivariate analysis adjusted for age, sex and study (model 2), and a multivariate analysis adjusted for all covariates (model 3). Only those participants who provided two or more waves of data were included in the joint longitudinal-survival analyses. Baseline Cox regression analyses were conducted

with Stata (StataCorp, 2007) and joint longitudinal-survival analyses were conducted with Mplus (Muthen & Muthen, 2007) statistical software.

Table 6.1 Baseline characteristics of survivors and decedents (N = 3,505).

	Survivors (n = 1987)		Deceased (n = 1518)	
	M	SD	M	SD
Continuous Variables				
Age	68.7	7.3	78.2	7.2
Hearing Threshold	23.4	13.7	34.4	15.1
Visual Acuity	0.8	0.2	0.6	0.3
MMSE	28.5	1.9	27.1	3.0
	N	%	N	%
Study				
BMES	1,552	78.3	430	21.7
ALSA	435	28.6	1,088	71.4
Sex				
Men	764	47.0	861	53.0
Women	1,223	65.1	657	35.0
Education				
Secondary only	831	50.7	809	49.3
Post-secondary, non-tertiary	919	64.2	513	35.8
Tertiary	148	61.4	93	38.6
Medical Conditions				
Stroke	87	57.6	64	42.4
Hypertension	710	57.5	524	42.5
Diabetes	120	47.6	132	52.4
Smoking				
Never	1,073	61.6	668	38.4
Former	749	51.4	709	48.6
Current	155	53.3	136	46.7

6.3 Results

The mean age of the sample was 72.8 years (range: 50-103) and 53.6% of the sample were female. There were 1518 participants who died during the study follow up period, their mean survival time was 6.7 years (SD = 3.9). Mortality data were right censored for 1987 participants, who were followed for an average of 11.0 years (SD = 2.3). At baseline the mean hearing threshold for decedents was 34.4 dB (SD = 15.0), compared to 23.4 dB (SD = 13.7) for participants with censored survival times. There were 420 decedents and 130 survivors identified as having dual sensory loss.

Decedents were more likely to be older (OR = 1.17, SE = 0.00, p <.01), male, (OR = 1.93, SE = 0.18, p < .01), ALSA participants (OR = 4.75, SE = 0.42, p < .01), and be current (OR = 3.01, SE = 0.52, p < .01) or former smokers (OR = 1.43, SE = 0.14, p < .01). Age and sex adjusted linear mixed models revealed that decedents had higher initial hearing thresholds (β = 3.1, SE = 0.52, p < .01) and greater annual rates of change in hearing thresholds (β = 0.12, SE = 0.05, p = .04) compared to survivors, who did not die during the study follow-up period. A total of 948 participants were excluded from the joint longitudinal survival analyses as they provided only one wave of data. Participants who dropped out before the second wave of data collection were older and had higher hearing thresholds compared to participants who met the criteria for inclusion in the joint parameter growth-survival model (n = 2557).

6.3.1 Testing Proportional Hazards Assumption

Plotting the scaled Schoenfeld residuals against survival time (Figure 6.2) did not provide strong evidence that the proportional hazard assumption was violated for hearing thresholds. Statistical test of the proportional hazard assumption further supported this interpretation (rho = 0.05, χ^2 = 3.21, p = .07).

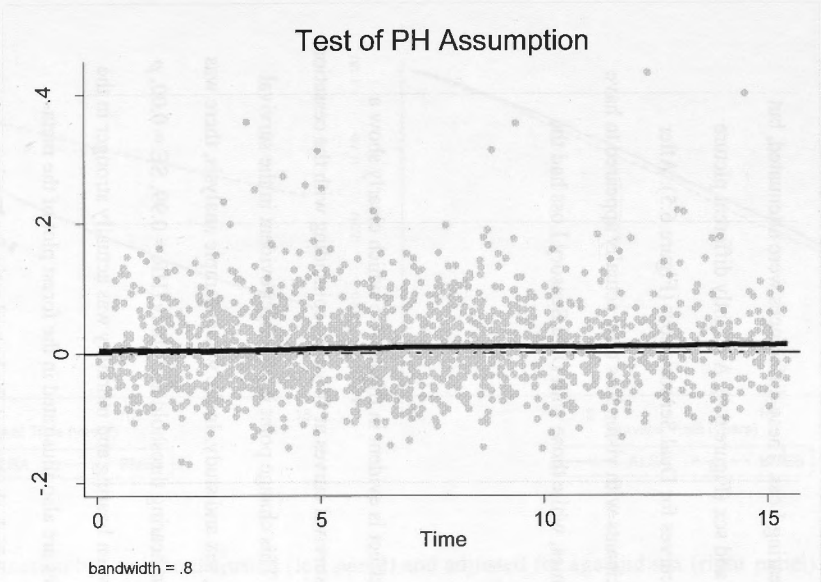


Figure 6.2 Partial residual plot for hearing thresholds. A lowess smoothing line parallel to the y-origin indicates that the proportional hazards assumption is satisfied. $\chi^2 = 3.21$, $p = .07$.

6.3.2 Kaplan-Meier Survival Curves

The Kaplan-Meier survival curves in Figure 6.3 show that there was a higher mortality rate in ALSA than BMES; however this was explained by study differences in age and sex distributions. Participants with mild levels of Hearing Loss and moderate to severe levels of Hearing Loss at baseline had shorter survival times compared to participants with no baseline Hearing Loss. These differences were attenuated, but remained after adjusted for age and sex (Figure 6.4). A slightly different picture emerged for the Kaplan-Meier curves for Dual Sensory Loss (Figure 6.5). After adjusting for age and sex, participants with vision loss only actually appeared to have the longest expected survival times, while those with Dual Sensory Loss had the shortest expected survival time.

6.3.3 Study Effects

The existence of a study effect is evident in figures 4-5 which clearly show a change point in the unadjusted survival curves at 10 years, coinciding with the cessation of the BMES follow up period. This change point is no longer evident in the survival curves that are adjusted for age, sex and study. In the whole sample analysis, there was a significant interaction between hearing threshold and study (HR = 0.99, SE = 0.00, p = .045) suggesting the link between hearing and mortality was actually stronger in the BMES sample. Study differences are also illustrated in the forest plot of the meta-analysis depicted in Figure 6.6, which shows that the hazard ratio for hearing thresholds was significant for BMES but not for ALSA. ALSA also had a smaller confidence interval, and therefore greater weighted contribution to the pooled estimate. Regardless, the pooled estimate for hearing thresholds was marginally statistically significant (Pooled Estimate = 1.04, 95% CI: 1.00-1.09, p = .04). Perhaps reflecting the reduced power due to smaller numbers, the study interactions were not significant in the sex specific analyses. Overall, there were mainly trivial differences in the effect sizes

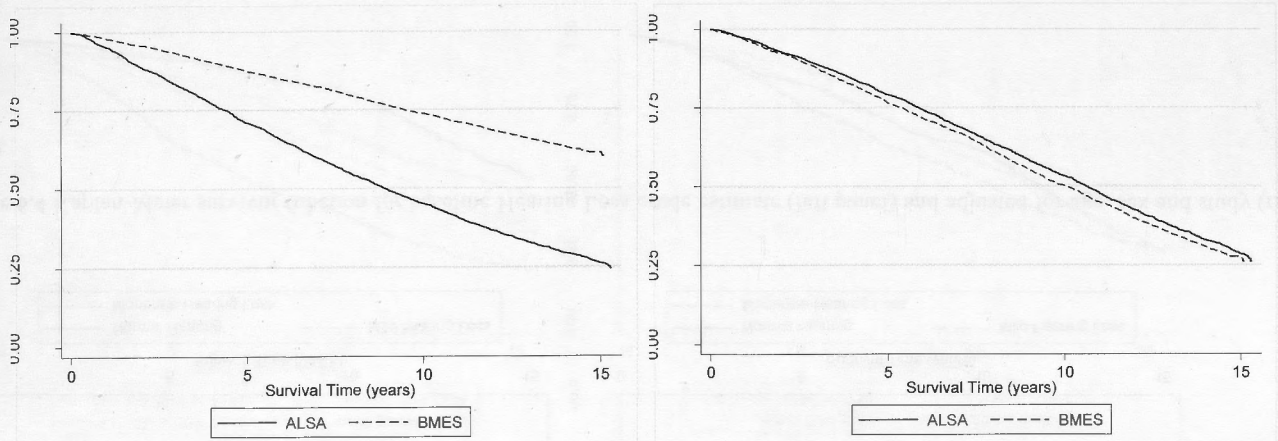


Figure 6.3 Kaplan-Meier survivor function by study, unadjusted (left panel) and adjusted for age and sex (right panel).

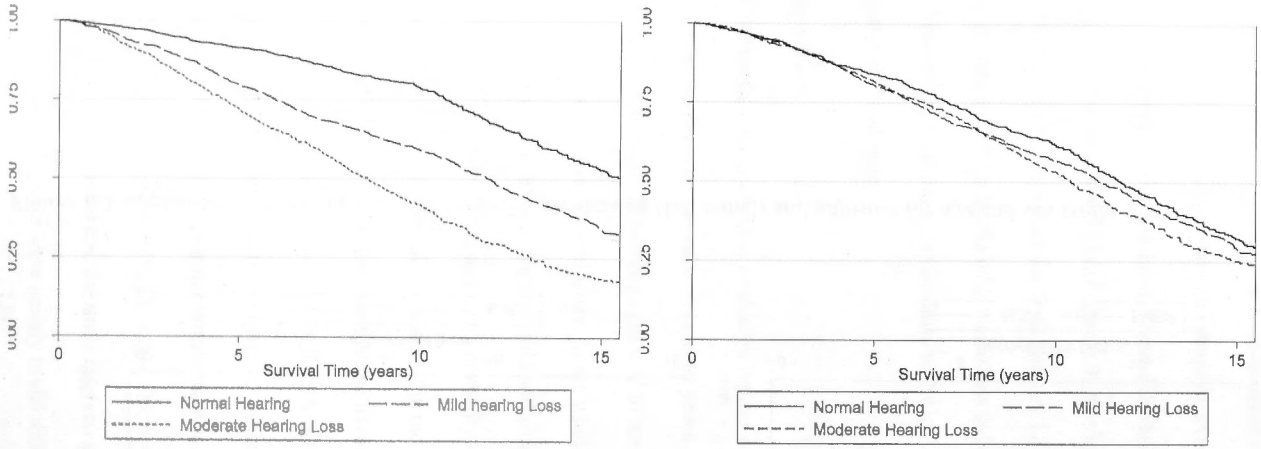


Figure 6.4 Kaplan-Meier survivor function for baseline Hearing Loss crude estimate (left panel) and adjusted for age, sex and study (right panel).

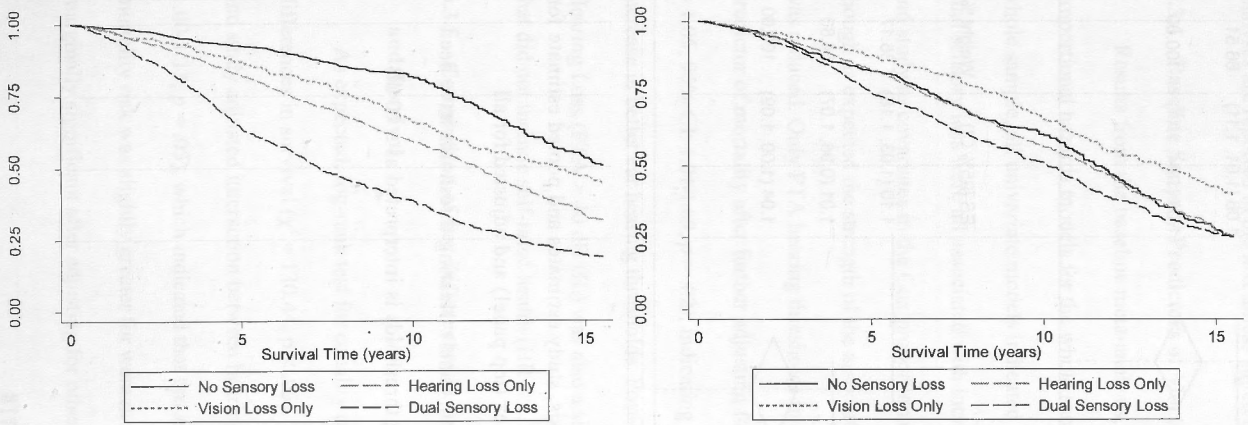


Figure 6.5 Kaplan-Meier survivor function for baseline Dual Sensory Loss crude estimate (left panel) and adjusted for age, sex and study (right panel).

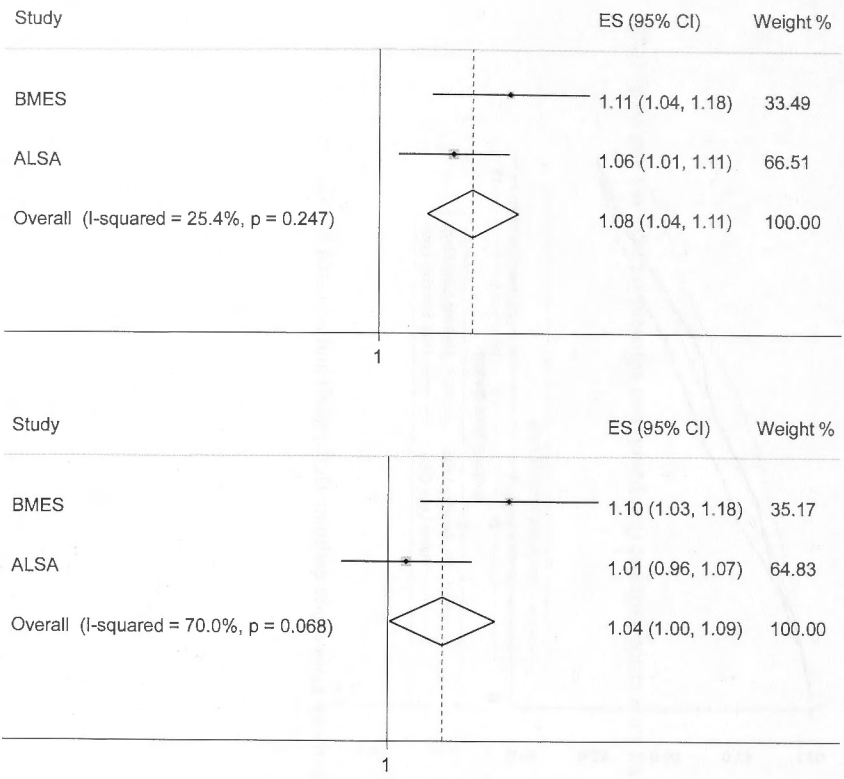


Figure 6.6 Forest plots from meta-analysis, study estimates and pooled estimate for hearing thresholds adjusted for age and sex (top panel) and adjusted for all demographic and health covariates (bottom panel). Hearing thresholds have been rescaled so a one unit increase in hearing thresholds is interpreted as a 10 dB increase in PTA.

and significance levels, estimated by the meta-analysis, study stratified analyses and study adjusted analyses. Due to the negligible variability between estimates provided by these three approaches to addressing study effects, estimates from the study adjusted analyses are reported in the text unless stated otherwise.

6.3.4 Baseline Sensory Predictors of Mortality for the Whole Sample

Results from the baseline meta-analysis, study stratified and study adjusted Cox proportional hazards models for the whole sample are presented in **Table 6.2**. For the whole sample, all univariate models indicated that hearing thresholds, hearing loss, and dual sensory loss were all associated with increased mortality risk. Inclusion of age, sex and study as covariates in the Cox proportional hazards model did not alter this finding, though as expected the strength of the association between hearing loss and mortality was reduced. Only PTA hearing thresholds in the better ear remained a reliable predictor of mortality after further adjusting for demographic and health covariates (HR = 1.05, 95% CI: 1.01-1.09, $p = .02$), indicating a 5% increased mortality risk for a 10 dB increase in better ear hearing thresholds. Post-hoc analyses revealed that moderate Hearing Loss (PTA > 40 dB HL) was also a significant predictor of mortality in models that did not include self-rated health (HR = 1.19, SE = 0.10, $p = .04$).

6.3.5 Comparison of Sex Differences in Baseline Associations between Hearing and Survival

As expected, log-rank test for equality of survivor function revealed sex differences in survival ($\chi^2 = 130.44$, $p < .05$). There was a marginally significant age and study adjusted interaction between hearing threshold and sex (HR = 1.05, 95% CI: 1.00-1.13, $p = .05$), which indicated that the association between hearing thresholds and mortality risk was slightly greater for women than for men. This interaction remained marginally significant after adjusting for other covariates in the full model (HR = 1.08,

95% CI: 1.02-1.16, p = .04). There was also an interaction between sex and dual sensory loss in the full multivariate model (HR = 1.69, 95% CI: 1.24-2.32, $p < .01$), again indicating stronger associations among women.

These sex differences in hearing survival relations were also evident in the analyses conducted separately for men (**Table 6.3**) and women (**Table 6.4**). In unadjusted models, crude hazard ratios for hearing loss and dual sensory loss were significant for both men and women, though the strength of the association was greater for women. Inclusion of age and study in the second set of models further revealed a divergent pattern of results between men and women. While both hearing thresholds and Hearing Loss (mild and moderate) were reliable predictors of mortality for women, only hearing thresholds were so for men. Finally, the full model revealed no unique association between hearing and mortality amongst men. In contrast, both Dual Sensory Loss (HR = 1.40, SE = 0.19, $p = .01$) and hearing thresholds (HR = 1.07, SE = 0.03, $p = .03$) significant predictors of mortality amongst women, whereas mild and moderate Hearing Loss were not.

6.3.6 Main Effects and Interaction Terms for Age, MMSE and Visual Acuity

There was a significant main effect for MMSE<24 indicating that participants with low cognitive function were at increased mortality risk (HR = 1.31, 95% CI: 1.07-1.59, $p < .01$). The main effect for visual acuity did suggest that adults with poor vision were at increased mortality risk though this was not statistically significant (HR = 0.88, 95% CI: 0.69-1.12, $p = .32$). There were no significant interactions between hearing thresholds and age in the whole sample or sex specific analyses, either before or after adjusting for other covariates. There were no significant interactions between hearing thresholds (PTA) and visual acuity or MMSE < 24.

Although it was not the focus of this study, post hoc analyses with visual acuity as the primary independent variable of interest were also conducted. After covarying the

effects of age and study, there were significant associations between poor visual acuity and mortality for women (HR = 0.63, 95% CI: 0.44-0.89, $p = .01$) but not for men (HR = 0.77, 95% CI: 0.56-1.03, $p = .08$). In the sex specific full multivariate models this pattern remained with stronger associations for women (HR = 0.78, 95% CI: 0.53-1.13, $p = .19$) than men (HR = 0.99, 95% CI: 0.72-1.35, $p = .94$).

6.3.7 Dual Sensory Loss

There were no significant main effects for Dual Sensory Loss for the whole sample when adjusting for demographic and health covariates. However, clear sex differences in dual sensory-mortality associations emerged when analyses were split for men and women. Among women, Dual Sensory Loss was independently associated with significantly increased mortality risk (HR = 1.40, 95% CI: 1.08-1.81, $p = .01$). Women with Vision Loss only or Hearing Loss only also had shorter survival times compared to women with no sensory loss, though these differences were not reliable for $p < .05$. In contrast, Dual Sensory Loss was not a significant predictor of increased mortality risk among men. Surprisingly, men with Vision Loss only actually had longer survival times relative to men with no sensory loss (HR = 0.62, SE = 0.10, $p < .01$). It is notable that this effect was in the opposite direction and not significant prior to adjusting for age (HR = 1.31, SE = 0.20, $p = .07$). This is an example of Lord's paradox (Tu, Gunnell, & Gilthorpe, 2008), as the effect of a categorical variable (vision loss only) on a continuous outcome (survival time) was reversed with the inclusion of a third continuous independent variable (age). This is illustrated in the Kaplan-Meier curve for Dual Sensory Loss (Figure 6.5).

Table 6.2 Main effect Hazard Ratios for hearing thresholds, hearing loss and dual sensory loss for the whole sample with study effects accounted for by meta-analysis, stratified analysis or adjusted analysis.

	Pooled Estimate			Stratified by Study			Adjusted for Study		
	Estimate	**[95% CI]**	***p***	**Estimate**	**[95% CI]**	***p***	**Estimate**	**[95% CI]**	***p***
Age and Sex Adjusted									
Hearing Thresholds	1.08	[1.04, 1.11]	<.01	1.08	[1.04, 1.11]	.00	1.07	[1.04, 1.11]	<.01
No Hearing Loss	reference								
Mild Hearing Loss	1.20	[1.06, 1.36]	.01	1.20	[1.06, 1.36]	.00	1.20	[1.06, 1.36]	<.01
Moderate Hearing Loss	1.28	[1.11, 1.48]	<.01	1.28	[1.11, 1.48]	.00	1.27	[1.1, 1.47]	<.01
No Sensory Loss	reference								
Hearing Loss Only	1.16	[1.01, 1.34]	.04	1.17	[1.02, 1.35]	.03	1.17	[1.02, 1.34]	.03
Vision Loss Only	0.92	[0.74, 1.14]	.43	0.96	[0.78, 1.19]	.73	0.96	[0.78, 1.18]	.71
Dual Sensory Loss	1.34	[1.14, 1.58]	<.01	1.31	[1.12, 1.54]	<.01	1.31	[1.11, 1.54]	<.01
Full Model									
Hearing Thresholds	1.04	[1.00, 1.09]	.04	1.05	[1.01, 1.09]	.02	1.05	[1.01, 1.09]	.02
No Hearing Loss	reference								
Mild Hearing Loss	1.08	[0.94, 1.25]	.28	1.11	[0.97, 1.29]	.14	1.11	[0.96, 1.28]	.14
Moderate Hearing Loss	1.15	[0.98, 1.36]	.09	1.17	[0.99, 1.38]	.06	1.17	[0.99, 1.38]	.06
No Sensory Loss	reference								
Hearing Loss Only	1.09	[0.94, 1.26]	.27	1.11	[0.96, 1.29]	.17	1.11	[0.95, 1.28]	.19
Vision Loss Only	0.83	[0.66, 1.04]	.10	0.85	[0.62, 1.17]	.33	0.87	[0.70, 1.09]	.22
Dual Sensory Loss	1.09	[0.91, 1.30]	.36	1.04	[0.82, 1.33]	.73	1.07	[0.90, 1.28]	.43

Note: Full Model adjusted for age, sex, education, visual acuity, MMSE<24, smoking status (current and former), self-rated health, diabetes, hypertension, and stroke.

Hearing thresholds were PTA in the better ear divided by 10 to aid estimation and interpretation of small estimates.

Table 6.3 Main effect Hazard Ratios for hearing thresholds, hearing loss and dual sensory loss for men with study effects accounted for by meta-analysis, stratified analyses or adjusted analysis.

	Pooled Estimate			Stratified by Study			Adjusted for study		
	Estimate	[95% CI]	*p*	Estimate	[95% CI]	*p*	Estimate	[95% CI]	*p*
Age Adjusted									
Hearing Thresholds	1.06	[1.01, 1.11]	.02	1.06	[1.01, 1.11]	.02	1.06	[1.01, 1.11]	.02
No Hearing Loss	reference								
Mild Hearing Loss	1.16	[0.98, 1.38]	.08	1.16	[0.98, 1.38]	.08	1.16	[0.98, 1.38]	.08
Moderate Hearing Loss	1.20	[0.99, 1.45]	.07	1.21	[1.00, 1.46]	.06	1.20	[0.99, 1.46]	.06
No Sensory Loss	reference								
Hearing Loss Only	1.04	[0.86, 1.25]	.70	1.06	[0.88, 1.27]	.57	1.05	[0.87, 1.27]	.58
Vision Loss Only	0.64	[0.47, 0.88]	.01	0.71	[0.52, 0.96]	.03	0.71	[0.52, 0.96]	.03
Dual Sensory Loss	1.05	[0.84, 1.31]	.66	1.06	[0.85, 1.32]	.61	1.06	[0.85, 1.32]	.62
Full Model									
Hearing Thresholds	1.03	[0.98, 1.09]	.28	1.04	[0.99, 1.10]	.16	1.04	[0.99, 1.10]	.16
No Hearing Loss	reference								
Mild Hearing Loss	1.02	[0.84, 1.25]	.82	1.08	[0.89, 1.31]	.46	1.08	[0.89, 1.31]	.45
Moderate Hearing Loss	1.10	[0.88, 1.37]	.40	1.15	[0.92, 1.43]	.22	1.15	[0.92, 1.43]	.22
No Sensory Loss	reference								
Hearing Loss Only	0.98	[0.80, 1.19]	.82	1.01	[0.83, 1.23]	.95	1.01	[0.83, 1.23]	.94
Vision Loss Only	0.55	[0.39, 0.77]	<.01	0.62	[0.45, 0.86]	.00	0.62	[0.45, 0.86]	<.01
Dual Sensory Loss	0.82	[0.64, 1.04]	.10	0.86	[0.68, 1.08]	.20	0.86	[0.68, 1.08]	.20

Table 6.4 Main effect Hazard Ratios for hearing thresholds, hearing loss and dual sensory loss for women with study effects accounted for by meta-analysis, stratified analyses or adjusted analysis.

	Pooled Estimate			Stratified by Study			Adjusted for study		
	Estimate	**[95% CI]**	***p***	**Estimate**	**[95% CI]**	***p***	**Estimate**	**[95% CI]**	***p***
Age Adjusted									
Hearing Thresholds	1.11	[1.05, 1.17]	<.01	1.10	[1.05, 1.16]	<.01	1.10	[1.04, 1.16]	<.01
No Hearing Loss	reference								
Mild Hearing Loss	1.23	[1.02, 1.48]	.03	1.24	[1.03, 1.49]	.02	1.24	[1.03, 1.49]	.02
Moderate Hearing Loss	1.39	[1.12, 1.73]	<.01	1.38	[1.11, 1.72]	<.01	1.37	[1.10, 1.70]	.01
No Sensory Loss	reference								
Hearing Loss Only	1.28	[1.04, 1.58]	.02	1.28	[1.04, 1.58]	.02	1.28	[1.03, 1.57]	.02
Vision Loss Only	1.28	[0.95, 1.72]	.10	1.31	[0.98, 1.74]	.07	1.30	[0.98, 1.74]	.07
Dual Sensory Loss	1.72	[1.35, 2.19]	<.01	1.70	[1.33, 2.16]	<.01	1.68	[1.32, 2.14]	<.01
Full Model									
Hearing Thresholds	1.07	[1.01, 1.15]	.04	1.07	[1.01, 1.15]	.03	1.07	[1.01, 1.15]	.03
No Hearing Loss	reference								
Mild Hearing Loss	1.10	[0.89, 1.37]	.38	1.12	[0.90, 1.38]	.32	1.11	[0.90, 1.38]	.33
Moderate Hearing Loss	1.20	[0.93, 1.55]	.16	1.21	[0.94, 1.56]	.15	1.20	[0.93, 1.55]	.16
No Sensory Loss	reference								
Hearing Loss Only	1.19	[0.95, 1.50]	.13	1.19	[0.95, 1.49]	.13	1.19	[0.95, 1.49]	.14
Vision Loss Only	1.30	[0.95, 1.78]	.11	1.25	[0.92, 1.70]	.15	1.26	[0.93, 1.70]	.14
Dual Sensory Loss	1.44	[1.11, 1.88]	.01	1.41	[1.08, 1.84]	.01	1.40	[1.08, 1.82]	.01

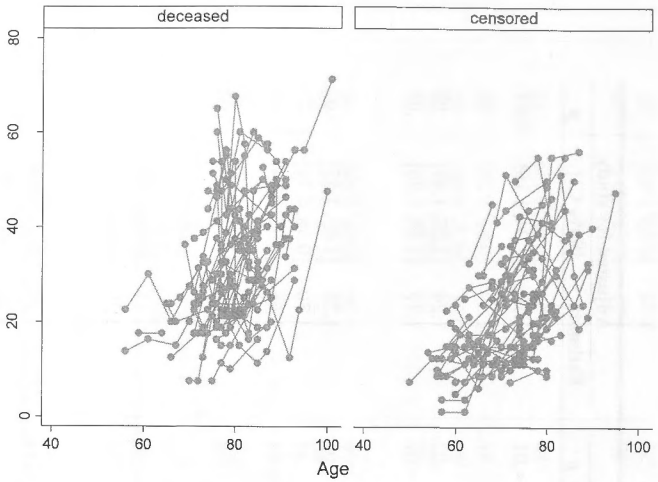


Figure 6.7 Raw longitudinal data for hearing thresholds for decedents and survivors (random selection of 10% of sample).

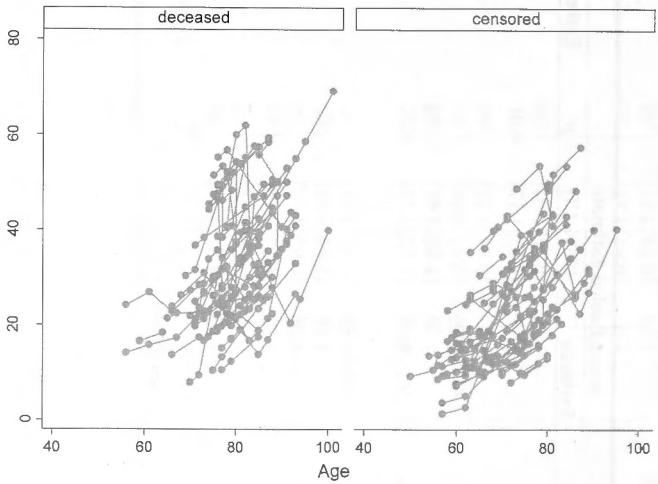


Figure 6.8 Predicted individual linear rates of change for hearing thresholds for decedents and survivors (random selection of 10% of sample).

6.3.8 Change in Hearing Thresholds Predicting Mortality Risk

Raw individual longitudinal hearing data for decedents and survivors are presented in **Figure 6.7**. Estimated individual linear trajectories of hearing thresholds for decedents and survivors are presented in **Figure 6.8**. From each of these figures it is possible to discern higher intercepts and steeper rates of change among decedents. As baseline analyses revealed negligible substantive differences between the three approaches to account for study effects, all model factors were residualized for study in the joint longitudinal-survival analysis. Of the fixed effects from the growth model component, the mean intercept was 29.30 dB HL (*SE* = 1.17) and the mean annual rate of change in PTA was 0.74 dB HL (*SE* = 0.11). Of the random effects, the variance for the intercept was 133.36 (*SE* = 8.20) and the variance for the slope factor was 0.27 (*SE* = 0.05). Table 6.5 shows the hazard ratios from the Cox survival component of the analyses. Unadjusted models revealed that hearing threshold intercepts and slopes were reliable predictors of mortality in the whole sample. In sex specific analyses, hearing intercepts were associated with mortality for both men and women, however rate of change in hearing was only associated with mortality for women. As in the case of the baseline analyses, adjusting for demographic and health covariates resulted in a significant effect of hearing threshold intercepts on survival time for women only. While there was a trend suggesting that participants observed to have faster rates of change in hearing were more likely to have shorter survival time, the confidence intervals were wide. Change in hearing was not a reliable predictor of mortality for men or women, independently of socio-demographics, health and initial hearing levels.

Table 6.5 Results from the Cox proportional hazards model component of the joint longitudinal-survival analysis (n = 2622), the sample was restricted to participants who provided a minimum of 2 waves of hearing data.

	PTA intercept			PTA change		
	HR	**[95% C.I]**	***p***	**HR**	**[95% C.I]**	***p***
Unadjusted model						
Women	1.03	[1.02, 1.04]	< .01	1.47	[1.06, 2.05]	.02
Men	1.03	[1.02, 1.03]	< .01	1.35	[0.92, 1.99]	.12
Whole Sample	1.03	[1.03, 1.04]	< .01	1.41	[1.11, 1.80]	.01
Age, sex and study adjusted model						
Women	1.01	[1.01, 1.02]	< .01	1.20	[0.90, 1.62]	.22
Men	1.01	[1.00, 1.01]	.13	1.19	[0.82, 1.72]	.37
Whole Sample	1.01	[1.01, 1.02]	< .01	1.20	[0.94, 1.53]	.14
Full Model						
Women	1.01	[1.00, 1.02]	.04	1.04	[0.73, 1.50]	.82
Men	1.01	[1.00, 1.01]	.13	1.19	[0.83, 2.41]	.37
Whole Sample	1.01	[1.00, 1.01]	.04	1.23	[0.89, 1.70]	.20

Note: The time metric is years in study.

HR: Hazard ratio

95% C.I: 95% Confidence Interval

6.4 Discussion

The main aims of the present study were to use pooled data from the DYNOPTA project to assess sex differences in associations between sensory loss and mortality. I evaluated whether categorization of hearing thresholds masks any independent links between hearing loss and increased mortality risk, and investigated if rate of change in hearing thresholds is an independent risk factor for mortality. The key findings regarding these three aims were that; 1) sensory loss in women was more strongly linked with increased mortality risk than in men, 2) hearing thresholds were more reliable predictors of mortality than polytimised hearing measures, and 3) rate of change in hearing was not an independent risk factor for mortality.

Although hearing thresholds were found to be a risk factor for mortality, the association was not strong and the underlying mechanism is not clear. It is not the contention of this chapter that poor peripheral hearing is a direct cause of death. Rather, one explanation is that age-related declines in hearing may be considered a bio-marker of biological and functional age (Anstey et al, 2001) that provides an alternative to chronological age as an index of how 'old' an individual is. Hearing loss and Dual Sensory Loss could be markers of illness or frailty, particularly among older women. Also, the link could be mediated by poor mental health which was not tested here. Loss of sensory function may result in increased social isolation, decreased capacity to adapt to changing circumstance, in turn can leading to depression, which has been recognised as a risk factor for mortality. Overall, I concur with Reuben et al (1999) that "it is probably reasonable to conclude that much of the effect of sensory impairment on mortality is caused by the co-morbid conditions that occur in older persons" (p.934).

It is possible that age-related sensory decline may be a stronger biomarker of functional age for women than for men. Previously, common factor loading between sensory-motor and cognitive domains have been reported to be greater in women than in

men (Christensen, Mackinnon, Korten, & Jorm, 2001), though this study used self-report measures of sensory loss. The opposite effect has been reported for grip-strength. Multivariate Cox regression models revealed that poor grip-strength was actually a stronger risk factor for mortality in men than in women (Al Snih, Markides, Ray, Ostir, & Goodwin, 2002). When investigating mortality differentials, it is important to consider how women's greater longevity may confound results if analyses are not conducted separately for men and women.

Despite offering grounds for a different conclusion concerning hearing-mortality relations, the results of the present study are consistent with previously published studies. When hearing was defined as a categorical variable reflecting ranges of hearing loss, in line with conventional practice in medical and epidemiological studies, there was no increased risk of mortality among men or women with mild or moderate degrees of hearing loss. Unlike previous studies, the primary analyses of interest presented here did not categorise participants into groups defined by percentiles (Anstey, Luszcz, Giles, et al., 2001) or standardised ranges of hearing thresholds. Although it is common practice in epidemiological research to dichotomize or polytimize continuous covariates to aid interpretation, it is well established that this reduces power and increases error variance (Babyak, 2004; MacCallum, Zhang, Preacher, & Rucker, 2002; Morgan & Elashoff, 1986; Royston, Altman, & Sauerbrei, 2006).

The approach by Anstey et al. (2001) to model hearing threshold quintiles with ALSA data was perhaps the closest approximation to modelling hearing thresholds as a linear variable. Again, the findings presented here do not contradict those findings. The study specific estimates used in the DYNOPTA meta-analysis indicated hearing was not a reliable predictor of mortality in ALSA. It was only after ALSA data was combined with BMES data, either through direct pooling of raw data or through pooling of estimates, that hearing thresholds were identified as independently associated with

mortality. However, unlike previous analyses of ALSA, decline in hearing was not shown to be related to shorter survival time. A number of explanations can account for this. Firstly, the present study used a larger sample of which ALSA comprised a subset, and more waves of data were available for analysis resulting in a later censoring date. Secondly, a different methodology was used whereby estimated individual hearing trajectories were tested as predictors of mortality whilst controlling for initial hearing levels. In contrast, Anstey et al. (2001) was restricted to using a binary variable of hearing change over two years and did not adjust for initial hearing levels.

The link between hearing, vision and dual sensory loss was clearly stronger for women. This is consistent with Lee et al. (2007) who found self-reported dual sensory loss was associated with mortality in women but not in men. The analyses presented in the present study not only replicate these findings, but extend them by demonstrating that they also apply to clinical measures of hearing thresholds and visual acuity. Lee et al. (2007) were unable to adjust for lifestyle behaviour and disease variables such as smoking status, diabetes or cardiovascular disease, but suggested that sensory-survival relations may be further explained by these factors. This hypothesis is not wholly supported in the present study; hearing remained an independent risk factor for women after adjusting for diabetes, hypertension, stroke and smoking.

This study applied recent developments in longitudinal analytic techniques to investigate associations between rates of decline in hearing thresholds with mortality by simultaneously fitting a latent growth curve within a survival model. Despite a trend which indicated that individuals with faster rates of hearing decline did have shorter survival times compared to the sample norm, the 11-year trajectories of hearing thresholds were not statistically reliable predictors of increased mortality risk after adjusting for potential confounds. This echoes research in cognitive ageing, which has similarly not identified cognitive decline rates as a risk-factor for mortality when using

either two stage or joint survival-growth modelling approaches (Batterham et al., 2011; Ghisletta, McArdle, & Lindenberger, 2006).

The importance of accounting for study design effects when analysing pooled data is again evident. ALSA has an older sample with a narrower age range compared to the BMES sample, which may explain the smaller variance and weaker association between hearing and mortality in ALSA. Importantly, there was little difference between the approaches to dealing with study effects. As expected, there was strong agreement between results derived from pooled estimates (i.e. meta-analytic approach) with results derived from pooling of raw data. This may have been due to the inclusion of only two studies. It would be interesting to evaluate how well findings converge when a larger number of studies are harmonised and pooled.

There are some limitations to the present study. Incomplete data may have biased findings from the full multivariate model, as 21% of the sample was dropped due to missing data. Unfortunately the version of Stata used does not support multiple imputation techniques for Cox regression models. Sensory functioning was restricted to pure-tone audiometry and visual acuity. It is acknowledged that there are other aspects of visual and auditory functioning which are likely to be important determinants of health, well-being and functional ability in older adults. One advantage measures of self-reported sensory loss have over objective measures is that when individuals make judgements on their perceived level of sensory impairment, they are likely to make a global assessment of their sensory capabilities. Also, the choice of time metric may have influenced our findings concerning rates of change in hearing predicting mortality. A maximum of 4 waves of data were available, and due to considerable attrition rates, models were restricted to testing for linear rates of change. This is unfortunate as rate of decline in hearing is known to accelerate with age.

CHAPTER 7: Cognitive, Health and Socio-demographic Predictors of Longitudinal Decline in Hearing Acuity among Older Adults

Synopsis

The aim of this chapter was to investigate predictors of rates of change in hearing thresholds in older adults. Linear mixed models tested for predictors of change in hearing. Hearing loss for high range frequencies preceded decline in low range frequencies. Men had higher baseline hearing thresholds, while women experienced faster rates of decline in hearing for mid to high range frequencies. The estimated rate of change for a 75 year old adult was 0.91 decibel hearing level (dB HL) per year for pure tone thresholds averaged over frequencies ranging between 0.5-4kHz in the better ear. Baseline age (β=0.03,p<.01), hypertension (β=0.15,p<.01), and probable cognitive impairment (β=0.40,p=.01) were independent predictors of annual rate of change in hearing thresholds. Incidence of probable cognitive impairment was also associated with higher hearing thresholds. Other known correlates for prevalence of hearing impairment, including low education, noise damage, diabetes, and history of stroke were independently associated with baseline levels of hearing, but were not predictive of change in hearing thresholds.

7.1 Background

Age-related hearing loss is highly prevalent among older adults (Gates & Mills, 2005; Lee, Gomez-Marin, Lam, & Zheng, 2004; Lin, Thorpe, et al., 2011; Yueh, Shapiro, MacLean, & Shekelle, 2003). It features among the leading causes of years lived with disability and is considered a substantial contributor to global burden of disease (Pascolini & Smith, 2009). Cross-sectional studies have identified diabetes(Austin et al., 2009; Cheng et al., 2009), cardiovascular disease, hypertension and blood pressure (Gates, Cobb, D'Agostino, & Wolf, 1993) as risk-factors for hearing loss. Hearing loss has also been linked with poor physical and mental health, falls (Viljanen et al., 2009), mortality(Anstey, Luszcz, Giles, et al., 2001; Karpa et al., 2010), and lower cognitive functioning or dementia (Gates, Anderson, Feeney, McCurry, & Larson, 2008; Gates, Beiser, Rees, D'Agostino, & Wolf, 2002; Lin, 2011b; Lin, Metter, et al., 2011; Tay et al., 2006; Uhlmann, Larson, Rees, Koepsell, & Duckert, 1989b; Wallhagen, Strawbridge, & Shema, 2008; Wingfield, Tun, & McCoy, 2005). However, longitudinal analyses have failed to show an association between many of these risk-factors with incidence of age-related hearing loss (Gopinath et al., 2010; Gopinath, Schneider, et al., 2009; Mitchell et al., 2009).

Divergent patterns of predictors for prevalence versus rates of decline in hearing have been suggested to arise from methodological factors. These include insufficient statistical power, differences in the rate of onset, and age dependency of hearing loss (Gopinath, Schneider, et al., 2009). Alternatively, the common practice of dividing ranges of averaged hearing thresholds into conventional categories of hearing loss (e.g. no impairment, mild impairment, moderate impairment) may obscure true associations between risk-factors for change in hearing acuity. This chapter addresses these issues by employing growth curve techniques to examine hearing trajectories in a larger representative sample of older adults than has previously been available. Other studies

investigating longitudinal changes in continuous measures have primarily focused on mapping age and sex trajectories of individual pure-tone frequencies (Brant & Fozard, 1990; Echt, Smith, Burridge, & Spiro III, 2010; Jane, Kathleen, & Robert, 1999; Lee, Matthews, Dubno, & Mills, 2005; Wiley et al., 2008). This study aims to extend the current understanding of age-related hearing loss by additionally investigating socio-demographic and health-related risk-factors for change in hearing thresholds[4].

2 This chapter was published in Kiely, K. M., Gopinath, B., Mitchell, P., Luszcz, M., & Anstey, K. J. (2012). Cognitive, Health, and Sociodemographic Predictors of Longitudinal Decline in Hearing Acuity Among Older Adults. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*. 67(9), 997-1003.

7.2 Methods

7.2.1 Participants

This study used participants who completed hearing assessments from the ALSA waves 1, 3, 6 and 7 and BMES waves 2 and 3.

7.2.2 Variables

Outcome variables used in analyses reported in this study were pure-tone thresholds in the better ear, and a pure-tone average (PTA) of low-to-mid range frequencies important for speech perception (0.5, 1, 2, 4kHz) in the better ear.

Medical conditions were obtained by self-report of clinician diagnoses and included: diabetes, hypertension, history of stroke and history of heart attack. Corrected visual acuity was tested with a logMAR chart at a distance of 3 meters, with visual impairment defined by values greater than 0.3 logMAR. A score of 23 or less on the Mini Mental State Examination (MMSE) (Folstein et al., 1975a) was used as an indicator of probable cognitive impairment. Smoking status was also obtained by self-report. Audiometric noise notches and self-reported occupational noise exposure (5+ years) were included to adjust for probable noise damage.

7.2.3 Analyses

For descriptive purposes the mean and standard deviation PTA were calculated for 10-year age-groups and for each covariate. Linear mixed models were used to estimate trajectories of hearing thresholds in the better ear. All analyses included random effect variance components for the intercept and slope (time) with an unstructured covariance matrix. The optimal scaling of time was ascertained by comparing Bayesian Information Criterion (BIC) values for models that indexed time as linear and quadratic functions of age, with models that indexed time over a 'years in

study' metric adjusting for age at baseline with an interaction term between age at baseline and 'years in study'. Better model fit is indicated by lower BIC values. Age and sex trajectories of hearing thresholds in the better ear were then estimated for each tone frequency and PTA. Model coefficients were used to graph the mean trajectories for men and women aged 60, 75 and 90 years at baseline. The predicted mean ages at which the PTA trajectory crossed thresholds of 25 dB HL and 40 dB HL were estimated for men and women by solving the model equation for 'time'.

Interaction terms between baseline predictors and time tested between-person differences in hearing trajectories. Analyses included baseline predictors of age (mean centered to 75 years), sex (female=1) and indicators of probable cognitive impairment, diabetes, stroke, hypertension, visual impairment, and smoking status. Time invariant predictors were workplace noise exposure, high frequency audiometric noise notches, and socio-demographics. For those baseline conditions that were significantly associated with change in hearing thresholds, an indicator of post-baseline incidence was also included to test if incident medical conditions were also associated with hearing loss. A four stage procedure was employed to evaluate predictors of change in PTA. In the first stage, I conducted a series of univariate models that estimated unadjusted associations between each predictor variable with baseline hearing levels and longitudinal hearing trajectories. In the second step, I ran the same set of univariate models adjusting for age at baseline. I then estimated a full multivariate model that included all covariates. In the final step BIC were used to evaluate the multivariate model, which was refined by excluding model terms that did not contribute to the overall model fit. In order to determine the extent to which noise damage confounded inferences concerning age-related hearing loss, multivariate analyses were repeated excluding all participants who reported 5 years of workplace related noise exposure, or

were identified to have high frequency noise notches. All analyses were conducted using Stata version 10 (StataCorp, 2007).

Table 7.1 Baseline sample profile, 3526 Australian adults aged 50 and older.

			PTA (dB)	
	N	%	Mean	(SD)
Sex				
Men	1633	46.3	30.6	(15.7)
Women	1893	53.7	26.0	(14.7)
Age				
50-59	285	8.1	15.2	(11.3)
60-69	861	24.4	20.8	(13.4)
70-79	1562	44.3	28.7	(13.1)
80-89	750	21.3	38.5	(14.2)
90+	68	1.9	46.6	(17.0)
Hearing Loss				
normal	1718	48.7	16.0	(5.9)
mild	1140	32.3	32.4	(4.2)
moderate-severe	668	18.9	52.0	(11.8)
Qualification				
Secondary only	1647	46.7	29.7	(15.2)
Post-secondary	1442	40.9	26.5	(15.1)
Tertiary	242	6.9	25.9	(14.2)
Occupation				
Tradesperson	440	12.5	32.9	(17.1)
Plant, machine operators and drivers	129	3.7	31.6	(16.1)
Labourers and related workers	231	6.6	31.7	(16.1)
Other	2726	77.3	26.9	(14.7)
Smoking Status				
Never	1741	49.4	27.7	(15.7)
Former	1458	41.3	28.8	(14.8)
Current	291	8.3	26.9	(15.7)
Workplace Noise Exposure				
<1 year	2339	66.3	27.1	(14.8)
1 to 5 years	323	9.2	29.5	(16.0)
5+ years	864	24.5	30.6	(16.1)
Medical Conditions (self-report)				
Diabetes	252	7.1	31.4	(16.6)
Stroke	151	4.3	32.4	(16.3)
Heart attack	353	10.0	31.3	(15.3)
Hypertension	1234	35.0	27.4	(14.7)
Measured Conditions				
Visual Acuity>0.3 logMAR	507	14.4	35.0	(15.9)
MMSE<24	218	6.2	38.9	(16.7)

Note: **PTA** = Pure-tone Threshold Average (dB) of 0.5, 1, 2, 4 kHz in the better ear; **logMAR** = logarithm of the minimum angle of resolution. **SD** = standard deviation.

7.3 Results

7.3.1 Description of Sample Characteristics

The baseline sample profile is described in Table 7.1. The pooled sample comprised 4,221 participants (46.3% men) with a mean age of 73.6 years (Standard Deviation (SD) = 8.9, range = 50 to 103). A total of 366 participants were classified with probable cognitive impairment at baseline, with a further 274 incident cases in subsequent waves. There were 211 participants identified with high frequency audiometric noise notches at any time (mean baseline age = 69.9, 75.4% men) and 851 participants reported workplace related noise exposure for 5 or more years.

The average time intervals between successive waves were 3.8 (SD = 1.8), 6.1 (SD = 0.2) and 3.1 (SD = 0.2) years, with participants providing an average of 2 waves of data. Prior to the commencement of wave 2, 16.6% of participants were lost to attrition and a further 6.4% were deceased. The BMES sample (n = 2,334) only provided data for waves 1 and 2. Within the ALSA sample, 44.5% of baseline participants were deceased at wave 3, this increased to 58.8% at wave 4.

Audiometric testing was completed by 3,526 participants at baseline (PTA Mean (M) =28.2 dB, SD = 15.2), and 3,011 participants at wave 2 (M = 30.1 dB, SD = 15.5). Based on the ALSA sample, PTA data were available for 525 participants at wave 3 (M = 37.0 dB, SD = 14.3) and 391 participants at wave 4 (M = 38.6 dB, SD = 15.3).

7.3.2 Modelling of Time

Linear mixed models that indexed time over a 'years in study' metric and adjusted for baseline age (BIC = 54272.0) provided a better description of longitudinal change in PTA , and were preferable to models that indexed time using an 'age' metric (BIC = 55195.9). This was consistent with previous recommendations regarding the

optimal scaling of time in longitudinal analyses with broad age cohorts (Morrell et al., 2009). All subsequent results index time over a 'years in study' metric.

7.3.3 Trajectories of Hearing Thresholds for Men and Women

The estimated age-related trajectories for each of the seven pure-tone frequencies and PTA in men and women are presented in Figure 7.1. An increase in hearing thresholds over time indicates a decline in hearing acuity. Relative to high range frequencies, change in hearing thresholds for low range frequencies began later and accelerated with age. Age-related changes in frequencies greater than 4 kHz were observed for adults of all ages, whereas frequencies of 0.5 and 1 kHz did not show marked increases in pure-tone thresholds until individuals were aged in their 70s. There were no sex differences in rate of change in hearing for PTA and low range frequencies. However women had lower intercepts and faster increases in thresholds greater than 3 kHz. Sex differences in intercepts and slopes were greatest for mid-range frequencies. For adults aged 75 years old at baseline, the estimated mean PTA trajectory crossed a threshold of 25 DB HL (often defined as mild hearing impairment) at ages 67.8 years for males and 71.1 years for women. The estimated mean PTA trajectory crossed a threshold of 40 dB HL (often defined as moderate hearing impairment) at ages 83.2 years for men and 86.5 years for women.

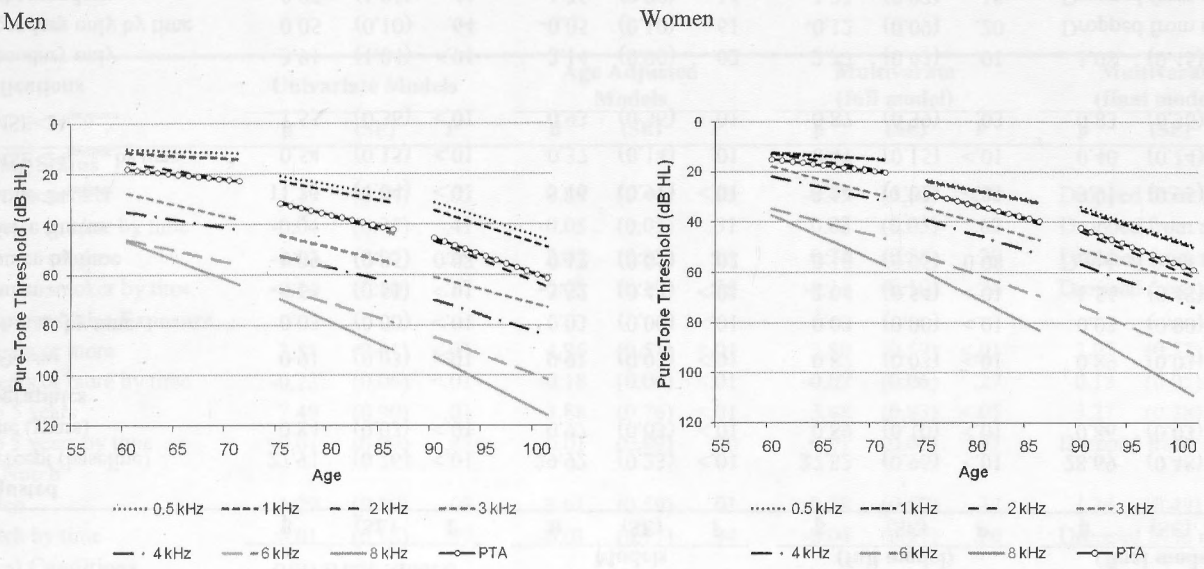


Figure 7.1 Unadjusted 11 year trajectories of pure-tone thresholds decibel hearing level (dB HL) for frequencies of 0.5, 1, 2, 3, 4, 6 and 8 kHz in the better ear, and PTA in the better ear, estimated for three cohorts of men (left panel) and women (right panel) aged 60, 75 and 90 years at baseline. The y-axis has been reversed so a negative gradient indicates a decline in hearing performance. Sample excludes participants with high frequency noise notches. The better ear was defined by PTA.

Table 7.2 Fixed effects for predictors of baseline levels and longitudinal trajectories of hearing thresholds (PTA) in the better ear, estimated from univariate and multivariate linear mixed models.

	Univariate Models			Age Adjusted Models			Multivarate (full model)			Multivarate (final model)		
	β	(SE)	P	B	(SE)	P	β	(SE)	P	β	(SE)	P
Unadjusted												
Intercept (baseline)	27.91	(0.26)	<.01	29.92	(0.23)	<.01	27.82	(0.96)	<.01	28.69	(0.48)	<.01
Time (years)	0.84	(0.03)	<.01	0.97	(0.03)	<.01	0.89	(0.10)	<.01	0.86	(0.03)	<.01
Demographics												
$Age_{baseline}$ †	0.91	(0.03)	<.01	0.91	(0.03)	<.01	0.87	(0.03)	<.01	0.89	(0.03)	<.01
$Age_{baseline}$ by time	0.03	(0.00)	<.01	0.03	(0.00)	<.01	0.03	(0.00)	<.01	0.03	(0.00)	<.01
Women	-4.64	(0.51)	<.01	-3.32	(0.44)	<.01	-2.04	(0.54)	<.01	-1.54	(0.48)	<.01
Women by time	0.09	(0.05)	0.07	0.12	(0.05)	.02	0.10	(0.05)	0.08	Dropped from model		
Cognitive Status												
$MMSE<24_{baseline}$	11.75	(1.04)	<.01	5.16	(0.92)	<.01	3.34	(1.02)	<.01	3.91	(0.95)	<.01
$MMSE<24_{baseline}$ by time	0.54	(0.15)	<.01	0.37	(0.14)	.01	0.47	(0.15)	<.01	0.40	(0.14)	.01
$MMSE<24_{incidence}$	1.55	(0.36)	<.01	0.93	(0.36)	.01	0.87	(0.39)	.03	0.83	(0.36)	.02
Qualifications												
Secondary only	3.91	(1.04)	<.01	2.14	(0.90)	.02	2.37	(0.93)	.01	1.08	(0.45)	.02
Secondary only by time	0.05	(0.10)	.64	-0.05	(0.10)	.61	-0.12	(0.09)	.20	Dropped from model		
Post secondary	0.87	(1.05)	.41	1.35	(0.90)	.14	1.23	(0.92)	.18	Dropped from model		
Post secondary by time	-0.02	(0.10)	.83	-0.04	(0.10)	.68	-0.08	(0.09)	.42	Dropped from model		

Table 7.2cont

	Univariate Models			Age Adjusted Models			Multivarate (full model)			Multivarate (final model)		
	β	(SE)	P	B	(SE)	P	β	(SE)	P	β	(SE)	P
Smoking Status												
Former smoker	1.19	(0.54)	.03	0.89	(0.46)	.05	-0.45	(0.51)	.38	Dropped from model		
Former smoker by time	-0.04	(0.05)	.41	-0.05	(0.05)	.31	0.03	(0.05)	.62	Dropped from model		
Current smoker	-1.03	(0.97)	.29	2.07	(0.83)	.01	0.24	(0.88)	.79	Dropped from model		
Current smoker by time	-0.14	(0.10)	.17	-0.03	(0.10)	.79	0.11	(0.10)	.24	Dropped from model		
Workplace Noise Exposure												
5 years or more	3.51	(0.61)	<.01	4.96	(0.51)	<.01	3.80	(0.59)	<.01	3.97	(0.57)	<.01
5 years or more by time	-0.23	(0.06)	<.01	-0.18	(0.06)	<.01	-0.07	(0.06)	.27	-0.13	(0.05)	.01
1 to 5 years	2.49	(0.90)	.01	3.88	(0.76)	<.01	3.48	(0.83)	<.01	3.27	(0.78)	<.01
1 to 5 years by time	-0.01	(0.09)	.87	0.01	(0.09)	.90	<.01	(0.08)	.97	Dropped from model		
Noise Notch												
Notch	1.29	(0.59)	.03	1.61	(0.59)	.01	0.78	(0.57)	.17	1.24	(0.49)	.01
Notch by time	-0.01	(0.18)	.97	-0.01	(0.17)	.94	-0.04	(0.17)	.80	Dropped from model		
Medical Conditions												
Hypertension	-1.38	(0.54)	.01	-0.93	(0.46)	.04	-0.77	(0.49)	.11	-0.79	(0.47)	.09
Hypertension by time	0.11	(0.05)	.04	0.10	(0.05)	.06	0.14	(0.05)	<.01	0.15	(0.05)	<.01
Diabetes	3.14	(1.01)	<.01	3.06	(0.86)	<.01	2.76	(1.14)	.02	2.09	(0.85)	.01
Diabetes by time	-0.09	(0.11)	.43	-0.06	(0.11)	.54	-0.23	(0.14)	.11	Dropped from model		
Stroke	4.67	(1.29)	<.01	3.28	(1.10)	<.01	2.66	(0.90)	<.01	2.56	(1.10)	.02
Stroke by time	-0.19	(0.16)	.22	-0.16	(0.15)	.29	-0.06	(0.10)	.56	Dropped from model		
Visual Impairment	8.66	(0.72)	<.01	2.04	(0.66)	<.01	1.31	(0.67)	.05	Dropped from model		
Visual Impairment by time	0.13	(0.08)	.08	-0.04	(0.08)	.59	-0.10	(0.07)	.15	Dropped from model		

Note: **MMSE<24$_{baseline}$** = Baseline Probable Cognitive Impairment; **MMSE<24$_{incidence}$** = Incidence of Probable Cognitive Impairment post baseline.

Random effects for intercept and slope are not shown.

†$Age_{baseline}$ is centered to 75 years.

Reference group for each variable: Male; No cognitive impairment; Tertiary qualified; Never smoker; Less than 1 year noise exposure; Absent noise notch; No reported hypertension; No reported diabetes; No reported stroke; and No visual impairment.

7.3.4 Predictors of Hearing Trajectories

Table 7.2 shows the results from the series of univariate, age and multivariate-adjusted linear mixed models for PTA in the better ear. In the age-adjusted univariate models, all baseline covariates reliably predicted initial levels of PTA. However, the only statistically significant predictors of rate of change were baseline age, sex, workplace noise exposure, and probable cognitive impairment. Faster increases in hearing thresholds were observed for older adults, women, and participants with probable cognitive impairment. Interestingly, noise notches were not associated with hearing trajectories but participants reporting five years or more of workplace noise exposure showed slower increases in hearing thresholds.

In multivariate analyses, smoking, visual impairment and post-secondary non-tertiary qualifications did not contribute to overall model fit and were excluded from the final model. For an adult aged 75 years old, the average PTA trajectory increased at a rate of 0.86 db HL per annum, with annual increase in the rate of change of 0.03 db HL. After adjusting for socio-demographic and health variables, there were no sex differences in rate of change in hearing, though probable cognitive impairment at baseline was associated with both poorer initial PTA levels (β_{level}=3.91, 95% CI=2.05-5.77) and faster rates of change in PTA (β_{change}=0.40, 95% CI=0.12-0.68). Incident probable cognitive impairment was also associated higher PTA ($\beta_{incident}$=0.83, 95% CI=0.12-1.55). Probable cognitive impairment at baseline was not associated with change in better ear thresholds for individual frequencies greater than 4kHz. Multivariate analyses also revealed greater rates of change in thresholds for participants reporting clinically diagnosed hypertension at baseline (β_{change}=0.15, 95% CI=0.06-0.25). Excluding participants who reported 5 years or more of workplace noise exposure or who had high-frequency noise notches, resulted in only minor adjustments to model coefficients and the substantive findings remained unchanged (data not shown).

7.4 Discussion

This study reports on patterns and predictors of change in 11-year trajectories for hearing thresholds in older adults. Hearing loss for frequencies important for speech perception increased at an average rate of 0.91 dB per year. Unsurprisingly, these rates of hearing decline were accelerated for older ages. Half of all adults in the oldest old cohort, aged 85 years and older, had moderate hearing loss, and almost all of the oldest-old cohort could be expected to have at least a mild degree of hearing loss. A key finding is that cognitive impairment was independently associated with lower levels and accelerated declines in peripheral hearing ability. Further, incidence of cognitive impairment was also associated with poorer hearing. Thus, both between-person differences and within-person change in cognitive function were identified as risk-factors for hearing loss. Hypertension was also found to be predictive of greater decline rates in hearing.

This study adds to the growing literature linking poor hearing with neurocognitive disorders (Gates et al., 2008; Gates et al., 2002; Lin, Metter, et al., 2011; Uhlmann et al., 1989b; Wallhagen et al., 2008; Wingfield et al., 2005) and age-related cognitive decline(Lin, 2011b). Early hearing loss and rapid hearing decline have been suggested to be precursors of dementia, and could be useful risk markers in dementia diagnosis (Gates et al., 2008; Lin, Metter, et al., 2011) though the analyses presented here do not test this hypothesis. Rather than assessing hearing loss as a leading indicator of cognitive decline, the model is specified such that individuals with cognitive impairment experience faster declines in peripheral hearing. That cognitive impairment was not predictive of decline in high frequency thresholds suggests underlying mechanistic pathways. However, the mechanism for this is unclear and cannot be identified from this study. The co-occurrence of cognitive impairment and hearing loss should be expected due to their associations with aging, but further explanation is

warranted because their association remains after statistically controlling for the effects of age. A third variable not properly adjusted for in this study, such as cerebral microangiopathy, is the most likely explanation for the association between cognition and hearing decline. As dementia pathology is not believed to affect the inner ear or cochlea (Sinha, Hollen, Rodriguez, & Miller, 1993), the current findings could simply be accounted for by top-down processing effects and reflect a more cautious or impaired decision making process regarding tone perception judgements. Older adults, particularly those with poor executive functioning, may show a response bias whereby greater certainty is required before they acknowledge an audible tone. To a lesser extent, these findings could partially be explained by difficulties experienced by people with sensory loss when completing standard neuropsychological assessments. However, such explanations can generally be discounted as it possible to conduct audiometric testing in young children, and trained clinical interviewers should be sensitive to hearing limitations of study participants(16).

A combination of histological, electrophysical and molecular mechanisms in both the peripheral and central nervous system underlie hearing loss (Van Eyken, Van Camp, & Van Laer, 2007). It is likely that any biological mechanism underlying a link between dementia and hearing loss occurs centrally, upstream of the cochlea (Gates et al., 2008). For example, Alzheimer Disease pathology has been observed in auditory system pathways such as the ventral nucleus of the medial geniculate body and in the auditory cortex, but not in cochlear nuclei (Sinha et al., 1993). As unaided pure-tone thresholds were used in this study, I am unable to draw direct inferences about the association between cognitive function and central auditory processing. Our understanding of the temporal inter-relations between hearing and cognition would be improved by longitudinal analyses of specific cognitive domains, hearing thresholds and hearing

measures that better asses central presbycusis and neural loss, such as dichotic listening or synthetic sentence identification tasks (Gates & Mills, 2005).

Our results support previous findings where risk-factors for prevalence of hearing loss, including smoking, diabetes and stroke (Gopinath et al., 2010; Gopinath, Schneider, et al., 2009; Mitchell et al., 2009), were not found to be predictive of incidence of hearing loss. Even cross-sectional associations between these factors and hearing loss remain in question. Recent analyses of 717 older adults in the National Health and Nutritional Examination Survey (Lin, Thorpe, et al., 2011) failed to find independent associations between low-frequency, speech-frequency, or high-frequency thresholds with the same set of risk-factors, regardless of whether thresholds were modelled as continuous or binary outcomes. This contrasts with our findings, as both diabetes and stroke were cross-sectionally associated with poor baseline hearing. These inconsistencies could arise from methodological differences and the larger sample available in DYNOPTA. Lin et al.(Lin, Thorpe, et al., 2011) also speculate that smoking, diabetes and other cardiovascular risk-factors may only have weak associations with hearing loss that are mediated or obscured by other factors. It is therefore intriguing to note the opposite pattern of results for hypertension, which was not predictive of baseline hearing levels but was a risk factor for change. The relation between hypertension and hearing loss is uncertain. Although some researchers have identified hypertension as being linked with hearing loss(2), in particular systolic blood pressure (Brant et al., 1996), this was not the case in the National Health and Nutritional Examination Survey (Lin, Thorpe, et al., 2011). This deserves further investigation.

Age-related declines in sensory functioning have multiple aetiologies, ranging from genetic factors (Wingfield et al., 2007) to environmental exposures (McMahon, Kifley, Rochtchina, Newall, & Mitchell, 2008; Van Eyken et al., 2007), but it has been argued recently that between-person differences in audiometric hearing thresholds can

be primarily attributed to genetic variation (Viljanen et al., 2007). If so, then this may explain why there has been a failure to show an association between changes in hearing performance with many of the known risk-factors for poor hearing. The inability to identify predictors for change in hearing and the equivocal cross-sectional findings suggest that rate of hearing decline may be a better indicator of putative normative or primary ageing processes and less influenced by disease than other functions. If higher intercepts reflect earlier onset of decline, this could indicate that hearing loss may begin at earlier ages for individuals with poor health, but the rate of hearing loss remains stable for most groups, with the exception of individuals with cognitive impairment or hypertension.

Paradoxically, there was no evidence of a relation between audiograms indicative of noise damage with hearing trajectories, yet noise exposure was predictive of more gradual declines in hearing. This is not completely inconsistent with a previous study that demonstrated slower hearing change for frequencies between 3-6kHz, yet accelerated change for adjacent frequencies of 2 and 8 kHz, among individuals with noise notches (Gates, Schmid, Kujawa, Nam, & D'Agostino, 2000). These findings were based on a younger sample of males, and a different methodology to that employed in the current study. Our failure to identify high frequency noise notches as a risk factor for change could be due to the difficulty in reliably identifying notches in older adults, particularly for ages when noise induced hearing loss becomes concomitant with age-related hearing loss (Coles et al., 2000).

Our results are consistent with existing knowledge about the general progression of age-related hearing loss (Gates & Mills, 2005). Typically, age-related hearing loss begins with loss of the ability to perceive high frequencies then gradually extends to low range frequencies. High frequency hearing loss has previously been reported to begin during the 50s(Wiley et al., 2008), so it is likely that decline for high frequencies

began before study commencement. Although men had poorer hearing levels for mid and high range frequencies, women experienced faster rates of hearing decline for these ranges. The lower initial levels for men probably reflects an earlier age-onset of hearing loss.

Differential patterns of hearing loss occur across a spectrum of tone frequencies which can be either independent of, or related to age (Liu & Yan, 2007). Due to the time intervals between hearing measurements, I lacked the data to detect rapid declines that occurred independently of age effects over a short time frame. At least four distinct types of presbycusis have been classified, each characterized by a unique pattern of change (Van Eyken et al., 2007; Weinstein, 2000) which I was also unable to investigate here. This study has not included ototoxic agents (Van Eyken et al., 2007; Yueh et al., 2003) and genetic data were not available. Clinical diagnoses of dementia was also unavailable. These caveats notwithstanding, ours is the largest dataset to assess the predictors of hearing loss.

In summary, this study contributes to existing knowledge of the association between impaired cognitive function and hypertension with accelerated decline in hearing. Our findings highlight the need for researchers and clinicians to be aware of impaired cognitive functioning when assessing hearing performance, and conversely, of hearing limitations when diagnosing, screening and managing individuals with dementia or other cognitive impairments. With the projected rise in the age-adjusted prevalence of hearing loss, its relation to health, well-being and longevity, there is a need for greater awareness and a better understanding of the development of age-related hearing loss and its interaction with co-morbid chronic health conditions.

CHAPTER 8: Time Ordered Inter-associations between Audiometric Hearing Thresholds and Processing Speed in Older Adults

Synopsis

Hearing loss has been linked with cognitive dysfunction and is an independent risk factor for incident dementia. However, it is unknown if the temporal precedence of hearing loss applies to fluid cognitive abilities such as processing speed. The aims of this study were to test time-ordered inter-associations between uncorrected pure tone hearing thresholds and processing speed using dynamic longitudinal modelling techniques. Data from the Australian Longitudinal Study of Ageing were analysed using Bivariate Dual Change Score models. The sample comprised 1172 community dwelling adults aged between 65 and 98 years (mean age= 73, male=52%) who completed cognitive and audiometric clinical assessments on up to four measurement occasions over a period of up to 12 years. Hearing was assessed by uncorrected pure-tone thresholds averaged across frequencies of 0.5, 1, 2, 4 kHz in the better ear. Processing speed was assessed by the Digit Symbol Substitution test. Results revealed a unilateral coupling pattern whereby higher hearing thresholds (poorer hearing) predicted faster rates of decline in processing speed over annualized time lags. Processing speed was not found to be a leading indicator of declines in hearing thresholds. These findings support the supposition that peripheral hearing loss is an early marker of subsequent cognitive

decline. Mechanisms that may explain why hearing loss is an antecedent of cognitive decline are discussed.

8.1 Background

Understanding structural relations between functional domains is an important step to developing a comprehensive explanation of human lifespan development. Cognitive and sensory functioning are two broad ability domains that are central to the study of human ageing, and also demonstrate the importance of considering cross-domain interdependencies in human development. As discussed in Chapter 1, neurocognitive disorders and sensory disorders are the two leading contributors to years lived with disability in adults aged 65 years and older (Alzheimer's Disease International, 2009; Australian Institute of Health and Welfare, 2003) with hearing loss in particular, considered one of the most prevalent chronic condition amongst older adults (Cruickshanks et al., 1998). The co-occurrence of sensory and cognitive losses may compound their respective impacts on adult health and well-being by placing constraints on available coping resources, therefore limiting an individual's capacity to adapt to changing circumstances. The focus of the present paper is not on the range of adverse outcomes that result from age-related decline in cognition and hearing, but to elucidate the reciprocal effects each has upon the other.

It has long been recognized that cognition and sensation do not operate in isolation, rather they constitute a bidirectional duplex-like system in which top-down and bottom-up processes are intrinsically intertwined. In spite of this two-way interactive pathway linking these domains, our understanding of cognitive and sensory abilities is often informed by research that focuses on each domain independently. Over the past two decades a number of empirical studies demonstrating age-related associations between intelligence and sensation have led researchers interested in lifespan developmental psychology to explicitly reconsider how these domains change in concert throughout the ageing process (Anstey, 2008b; Anstey et al., 2003a; Baltes & Lindenberger, 1997; Hofer, Berg, & Era, 2003; Li & Lindenberger, 2002; Lindenberger

& Baltes, 1994; Murphy, Craik, Li, & Schneider, 2000; Rabbitt, 1991; Salthouse, Hancock, Meinz, & Hambrick, 1996; Schneider & Pichora-Fuller, 2000). These studies have investigated a number of sensory modalities not only including vision or hearing, but also olfaction (Dulay & Murphy, 2002), balance (Rabbitt et al., 2006) and touch (Li, Jordanova, & Lindenberger, 1998). In line with this, there has been a notable resurgence of interest and discussion concerning how to conceptualise the connection between age-related hearing loss and cognitive functioning within medical, psychological and epidemiological disciplines (Beck & Clark, 2009; Lin, 2011a). Much of this recent discussion that has fallen under the inter-disciplinary banner of Cognitive Hearing Science (Arlinger et al., 2009), which has recently begun a biennial conference in Linköping, Sweden (Linnaeus Centre HEAD, 2011). The present study contributes to this research agenda by evaluating temporally ordered connections between age-related declines in sensory and cognitive functioning.

8.1.1 Theoretical frameworks

Theories concerning the connection between sensory and cognitive ageing have followed two themes, the identification of explanatory causal mechanisms and the role of sensory abilities as indices of cognitive ageing. Baltes and Lindenberger (Baltes & Lindenberger, 1997; Lindenberger & Baltes, 1994) originally outlined four broad causal hypotheses proposed to conceptualise apparent age-related connections between sensory and cognitive systems. These are commonly referred to as common cause, sensory deprivation and degradation (or sensory underload), and cognitive load on perception. These theories have also been succinctly summarized by Schneider & Pichora-Fuller (Schneider & Pichora-Fuller, 2000) and later by Gallacher (2005) in a review focused exclusively on hearing.

The provocative common cause theory essentially appeals to a third variable, positing that a single mechanism or common biological aetiology is jointly responsible

for sensory and cognitive decline. While the parsimony of a common factor is attractive, this account has been controversial due to the shotgun approach to pinpoint the candidate cause, and the number of alternate and equally plausible interpretations of the supporting evidence. Proponents of sensory deprivation hypotheses maintain that long-standing and sustained degraded sensory inputs, such as adult onset hearing-loss, will affect central cognitive functions. This model predicts cascading bottom-up effects. A number of pathways potentially underpin this effect. This could be due to increased demands placed on a limited supply of cognitive resources which must be allocated to lower-level processing of degraded sensory information. Alternatively, sensory loss may result in decreased social engagement and lower levels of participation in stimulating activities which are important for maintaining or building cognitive reserve. This explanation is akin to a cascade model, which suggests that declines in sensory-motor functions may precede and indirectly influence declines in cognition. This is depicted in Figure 8.1, which shows how sensory loss can be the catalyst for a sequence of knock-on effects that ultimately lead reduced cognitive capacity. Conversely, explanations that appeal to cognitive load on perception make the opposite prediction to sensory underload models, predicting a cascade of top-down effects. They postulate that as cognitive processes exerting top-down control over perceptual systems are compromised, then perception of sensory inputs may become less sensitive (Gallacher, 2005).

Schneider and Pichora-Fuller (2000) offer two other models that contrast with common cause interpretations; a multiple cause model that attributes declines in cognition and sensory function to age-related domain specific processes, and a less modular perspective which characterises perception and cognition as integrated information processing systems that share available resources. The latter model recognizes functional and structural interconnections between sensation and cognition.

This can be considered a more nuanced and sophisticated model as it can accommodate common and multiple causes as well as top-down and bottom-up cascade models. As Schneider and Pichora-Fuller (2000, page 208) point out, there is no 'firewall' that exists between sensation, perception and cognition, and any boundary that seperates these domains will be highly porous.

An alternative view to that presented by Baltes and Lindenberger (1994) posits sensory-motor variables as functional biomarkers that index maturation processes of primary ageing (Birren & Cunningham, 1985). Under this interpretation, sensory function is considered to provide a more precise indicator of old age and central nervous system integrity than other indices such as chronological age (Anstey et al., 1993; Anstey, 1999; Anstey & Smith, 1999b; Li & Schmiedek, 2002; MacDonald, DeCarlo, & Dixon, 2011; MacDonald, Dixon, Cohen, & Hazlitt, 2004). Biomarker explanations suggest that age differences in cognitive function may be explained or mediated by sensory function. Consequently, the biomarker hypothesis has been argued to make similar predictions to an upward cascade model (Ghisletta & Lindenberger, 2005), whereby hearing is a leading indicator of subsequent changes in cognition. Of these accounts, generally the common factor, sensory deprivation and biomarker accounts have attracted the most discussion. It is important to acknowledge that none of these explanations are mutually exclusive and it is possible that a number of pathways underpin age-related associations between cognition and hearing (Anstey, et al 2003).

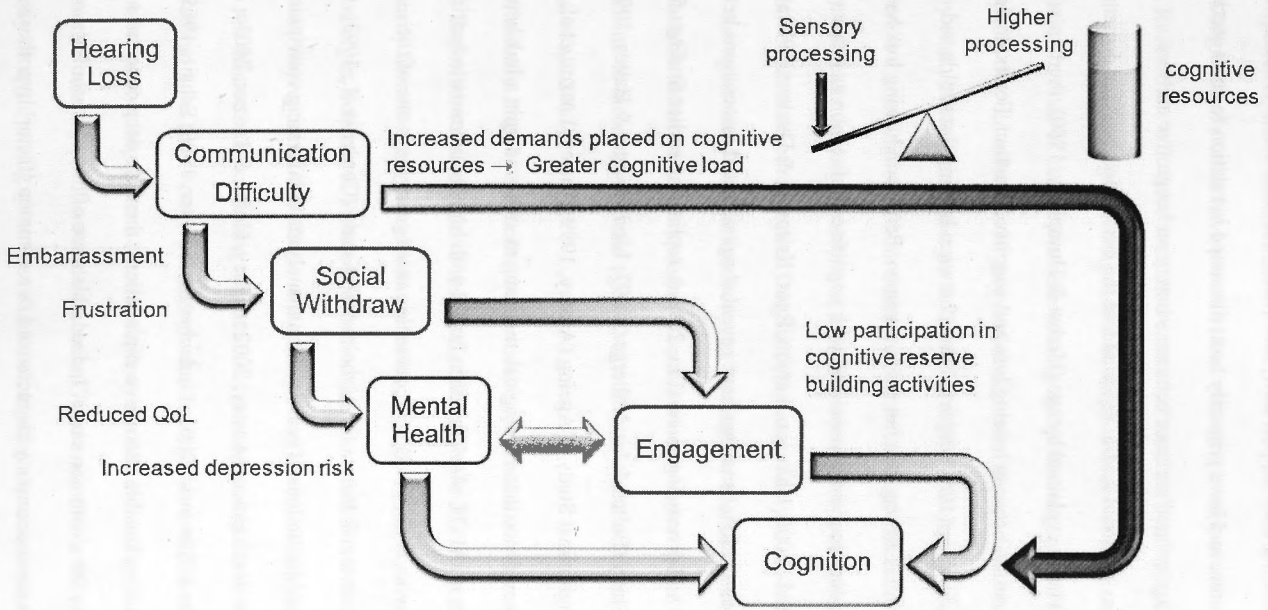


Figure 8.1 A bottom-up cascade model depicting two pathways between hearing loss and cognitive decline, one via a social mechanism (sensory deprivation) and the other via a resource allocation mechanism (sensory degradation).

8.1.2 Supporting Evidence

Although attracting little support now, historically common cause accounts were popular in the literature and have primarily been discussed in relation to cross sectional findings of shared age-related variance between sensory and cognitive abilities. Although early cross sectional studies reported that the association between dementia and deafness was wholly explained by age (Herbst & Humphrey, 1980), there is evidence of a connection between hearing loss and cognitive function. For example, Schaie, Baltes and Strother (1964) reported links between hearing thresholds and primary mental abilities among men but not for women. Peripheral hearing loss has been reported to be independently associated with cognitive dysfunction as measured by the MMSE (Tay et al., 2006; Uhlmann, Larson, Rees, Koepsell, & Duckert, 1989a). Further, recent cross-sectional analyses have revealed age adjusted associations between hearing thresholds and processing speed (Lin, 2011a), replicating earlier findings from the Berlin Aging Study (Baltes & Lindenberger, 1997; Lindenberger & Baltes, 1994) the Australian Longitudinal Study of Ageing (Anstey, 1999; Anstey, Luszcz, et al., 2001a). As further evidence that a biological mechanism may underpin a link between hearing and cognition, APOE e4/e4 has been linked with higher audiometric hearing thresholds (Kurniawan et al., 2012). Of course this same genotype is one of the strongest single genetic risk factors for Alzheimer's Disease (Corder et al., 1993). Given the well documented limitations of cross-sectional analyses in informing convincing theories of lifespan development (Anstey, 2002; Anstey, Hofer, & Luszcz, 2003c; Baltes, 1968; Hofer & Sliwinski, 2001; Lindenberger & Potter, 1998; Schaie, 1965) and the number of equally plausible alternative explanations, there are deep concerns and a sense of incredulity for a common cause. Indeed, this class of line of reasoning would perhaps have been more accurately characterised as a common 'factor' hypothesis as

this does not take the inferential leap of attributing covariation to an unobserved and poorly specified unitary mechanism.

Longitudinal studies have provided less evidence in support of a single common cause. Researchers who have evaluated common factor accounts of concomitant cognitive and sensory ageing using multivariate growth curves have reported little or no covariation in rates of change between hearing and processing speed. Importantly, any relationship that did exist was largely explained by health and other contextual variables (Anstey et al., 2003a; Lindenberger & Ghisletta, 2009). This lead to their conclusion that there is a combination of both domain general (common) and domain specific (multiple) factors driving age declines, a finding more easily accommodated by an integrative shared resource model. Despite being empirically under-identified, the notion that age-related associations between hearing and cognition can be attributed to a third variable (often loosely branded as a common neurological factor, a biological mechanism, or more simply brain ageing) is still entertained in the literature (Lin, Thorpe, et al., 2011).

Similarly, there is conflicting evidence in support of a sensory underload cascade model. Experimental studies have shown that degrading auditory inputs does not necessarily lower cognitive performance among healthy middle aged adults (Lindenberger, Scherer, & Baltes, 2001), though other studies have demonstrated such an effect (Surprenant, 2007). These study designs test the role of sensory degradation, but do not discredit the mediating role of social engagement in a cascade model. Longitudinal links have been reported between hearing loss and clinically significant levels of cognitive impairment. Central auditory dysfunction has been identified as a precursor of memory problems and dementia (Gates et al., 2011; Gates et al., 2002). This may be unsurprising given the reliance on top-down processes and executive functioning during central auditory tasks. For example, drawing on contextual cues to

understand degraded speech presented over competing background noise. Further, central auditory functions are more clearly dependent on the integrity of cortical structures and pathways. So despite the temporal sequencing, Gates' findings also conform to brain ageing explanations.

Peripheral hearing acuity that is subserved primarily by cochlear or inner ear functioning and measured by pure-tone audiometric thresholds, has also been reported to be independent risk factor for incident dementia (Lin, Metter, et al., 2011). An intriguing finding as dementia neuropathology are not generally considered to affect cochlear functioning (Sinha et al., 1993). This also raises the question, is hearing loss an antecedent of normative age-related cognitive decline, just as it is an antecedent of cognitive impairment or dementia. Such suppositions have prompted Lin (2011) to call for research investigating peripheral hearing as marker of non-dementia specific cognitive decline. On this the evidence swings both ways. After adjusting for baseline cognitive function, linear regression analysis of two waves of data from the Maastricht Ageing Study revealed low auditory acuity was predictive of greater difference scores for measures of recall, task shifting and learning (Valentijn, Boxtel, et al., 2005). Although Anstey, Luszcz and Sanchez (2001b) reported contrary findings with ALSA. Participants who experienced an increase in hearing thresholds of 10dB or more over two years did not have faster change scores in memory or processing speed when compared to participants whose hearing declined less than 10dB over the same period. However, hearing thresholds have not been tested as an early marker of cognitive decline by dynamic longitudinal modelling techniques.

In the case of hearing thresholds, intuitively it is less clear how cognition would be causative of poor peripheral hearing function as predicted by the cognitive load of perception hypothesis. Nevertheless, cognitive impairment, as measured by the MMSE, was reported to be an independent predictor of 11 year trajectories of uncorrected

hearing thresholds in Chapter 7 of this thesis (Kiely, Gopinath, Mitchell, Luszcz, & Anstey, 2012). It has been suggested that older adults with poor cognitive function may adopt a more conservative response bias and so may be reluctant to report the presence of a tone signal, or perhaps fail to report the presence of a tone due to reduced capacity for sustained attention (Lindenberger & Baltes, 1994).

8.1.3 The Present Study and Aims

There are both theoretical and empirical grounds for expecting dynamic longitudinal connections between hearing thresholds and cognition. Ghisletta and Lindenberger (Ghisletta & Lindenberger, 2005) conducted quadrivariate Dual Change Score Model (DCSM) analyses to evaluate lead lag associations between near visual acuity, distance visual acuity, processing speed and verbal knowledge. They found bidirectional links between age-related declines in sensory and cognitive functioning, specifically near vision and processing speed. Not only was near vision reported to be a leading indicator of decline in processing speed, but processing speed was equally a leading indicator of decline in near vision. On the basis of these results the authors concluded that a cascade hypothesis predicting that sensory-motor biomarkers to be catalysts for decline in cognition and intelligence, was not supported (Ghisletta & Lindenberger, 2005). It is not known if such temporally ordered and bidirectional coupling also generalises to other sensory-motor variables such as peripheral hearing acuity.

Dynamic longitudinal models, such as the DCSM allow testing of competing hypotheses concerning the direction of time-ordered associations linking distinct domains. Since its introduction (McArdle & Hamagami, 2001), use of the bivariate DCSM has become an increasingly common and accepted approach to test if preceding levels of functioning in one domain predict subsequent changes in another (Ferrer & McArdle, 2010). DCSMs have been used to assess lead-lag associations between

depressive symptoms and processing speed (Bielak, Gerstorf, Kiely, Anstey, & Luszcz, 2011), depression and memory (Jajodia & Borders), perceived control and health (Infurna, Gerstorf, & Zarit, 2011), functional limitations and memory (Infurna, Gerstorf, Ryan, & Smith), well-being and perceptual speed (Gerstorf, Lovden, Rocke, Smith, & Lindenberger, 2007) fluid and crystallized abilities (Finkel, Reynolds, McArdle, & Pedersen, 2007; Ghisletta & Lindenberger, 2003) and inter-relations within marital partners (Gerstorf, Hoppmann, & McArdle, 2008; Gerstorf, Hoppmann, Anstey, & Luszcz, 2009; Walker, Luszcz, Gerstorf, & Hoppmann, 2010).

It is important to take into account contextual variables when investigating cognitive ageing processes (Anstey, 2008a). Such contextual variables include education, mental health, health conditions and life style factors. For example, Anstey (2003) demonstrated that covariation between intercept and slope factors for hearing and a number of cognitive domains were largely explained by medical conditions, depression, and education. In light of this, the present analyses will take into account a number of shared risk factors for cognitive and hearing decline that may explain the association between these two domains. These include medical conditions such as diabetes, cardiovascular disease and stroke, and lifestyle factors (Gopinath et al., 2010; Gopinath, Schneider, et al., 2009). As evident in the previous Chapter 7, people identified with possible noise damage had higher hearing thresholds but more gradual rates of decline. The value of hearing as a biomarker for cognitive decline has previously been seen to be diminished due the confounding environmental factors like noise exposure (Anstey, 2008b). This study will therefore also account for noise induced hearing loss by adjusting analyses for self-reported occupational noise exposure. Finally, as dynamic links between vision and processing speed have been demonstrated, models will be adjusted for visual acuity.

The aims of this chapter are to extend our understanding of the longitudinal connection between sensory and cognitive functioning by testing time-ordered inter-associations between hearing acuity and processing speed. Four competing models were evaluated, namely 1) no dynamic coupling between hearing thresholds and processing speed, 2) a bi-directional coupling, such that both hearing thresholds and processing speed were equally leading indicators of change in the other, 3) a unidirectional relation where processing speed was a leading indicator of hearing decline only (consistent with a cognitive load hypothesis), and 4) a unidirectional relation where hearing thresholds were leading indicators of decline in processing speed only (consistent with the cascade, biomarker or sensory degradation hypotheses). While the DCSM cannot directly test the common cause hypothesis, it can be mapped onto predictions made by the sensory degradation and cognitive load hypotheses.

8.2 Methods

A series of bivariate dual change score models were fitted to 4 waves of 11 year longitudinal data from the Australian Longitudinal Study of Ageing (ALSA) (Luszcz et al., 2007). A detailed account of the overall study design and survey protocols was provided in Chapter 3, a brief description of details pertinent to the current study are provided below.

8.2.1 Sample

Included in these analyses were the 1172 participants who completed both audiometric and cognitive testing at baseline. The baseline characteristics are provided in Table 8.1.

8.2.2 Measures

Two outcome measures were PTA in the better ear and DSS score. To control for potential confounds, I adjusted models by covarying the effects of age at baseline (centered to 75years), sex (0=men, 1=female), age left school (0=age 14 or younger, 1=age 15 or older), occupational noise exposure (0=less than 5 years, 1=5 years or more), scores on the Mini Mental State Examination (MMSE) (Folstein, Folstein, & McHugh, 1975b), and Centre for Epidemiological Studies Depression Scale (CESD) (Radloff, 1977). Medical conditions (stroke, heart attack, hypertension) and visual acuity were also adjusted for.

For all analyses both PTA and DSS were transformed to a T-score metric (M=50, SD=10) standardized to the baseline ALSA sample as a reference group. Transforming the measures onto a common metric preserves the original variable properties, including longitudinal changes in means and variances, enabling direct comparisons of parameter estimates across scales and facilitating straightforward interpretation of results. For both hearing and processing speed the scale was orientated so higher values indicated better

performance and longitudinal trajectories with negative gradients indicated declining performance. All continuous covariates were mean centred.

8.2.3 Statistical Analysis

A comprehensive account of the specification, assumptions and limitations of the DCSM has been well documented over the past decade (Ferrer & McArdle, 2010; Jajodia, 2008; McArdle & Hamagami, 2001). DCSMs can be conceptualised as combining the properties of cross lagged panel regression with latent growth curves where longitudinal trajectories are partitioned into two distinct components of change, reflecting individual differences in change and population average in change. A schematic of the bivariate DCSM is presented in Figure 8.2, with X indicating PTA scores and Y indicating DSS scores. Following convention of structural equation model diagrams, squares indicate observed (manifest) variables, circles indicate unobserved (latent) factors, while fixed parameters are shown as one headed arrows and random parameters as two headed arrows. Unlabelled arrows are fixed to one. The triangle indicates that means and intercepts were estimated. Unmeasured 'node' variables were included on occasions where outcome variables were not assessed, this is a commonly applied step in specifying DCSMs to unbalanced panel data as it simplifies the model fitting procedure and ensures constant scaling across time allowing parameters to be interpreted here as annualized time lags (e.g. Bielak et al., 2011; Gerstorf et al., 2009; Walker et al., 2010).

A latent score at a given time t ($x_{[t]}$) is defined by the unit-weighted sum of latent score for the previous assessment ($x_{[t-1]}$) plus the latent difference score at time t ($\Delta x_{[t]}$). Intercept factors (x_0, y_0) represent baseline scores and slope factors (X_s, Y_s) represent linear change at the population level. All intercept and slope factors are allowed to vary (σ^2_{xo}, σ^2_{xs}, σ^2_{yo}, σ^2_{ys}) and covary (ρ_{xoxs}, ρ_{yoys}, ρ_{xoyo}, ρ_{xsys}, ρ_{xoys}, ρ_{xsyo}) while all error terms are assumed to be normally distributed with a mean of zero, constant variance

across time and uncorrelated with other model components. The latent difference scores ($\Delta x_{[t]}$) reflect reliable change between adjacent time points. These latent differences can be considered to be dependent variables in our analyses and are a function of three model components, which include 1) the within domain feedback effect of prior level on subsequent change given by auto-proportional β weights, 2) the within domain effect of the linear slope factor, and 3) the inter-domain cross-lagged γ weights. All β and γ parameters are constrained to be time-invariant within each domain. As the DCSM partials growth curve trajectories into two change components, namely the constant change slope factors (X_s, Y_s) and proportional change difference scores (β_x, β_y), neither should be interpreted independently of other model components. When the β and γ parameters are constrained to zero, the bivariate DCSM model is reduced to conceptually equivalent to, but an alternative specification of, a standard bivariate linear latent growth curve.

The primary foci of the modelling procedure are the coupling or cross-lag (γ) parameters, which regress latent difference scores for one domain ($\Delta x_{[t]}$) on prior levels of the coupled domain ($y_{[t-1]}$). In the context of the present study, $\gamma_{PTA \rightarrow \Delta DSS}$ is interpreted as the predictive effect of hearing on subsequent change in processing speed, conversely $\gamma_{DSS \rightarrow \Delta PTA}$ is interpreted as the predictive effect of processing speed on subsequent change in hearing. In an initial step, a full dynamics model is tested by allowing both cross lags to be freely estimated. By manipulating the inter-variable cross-lags in a series of models that are nested within the full dynamics model, we are able to make inferences concerning the directionality of dynamic coupling between domains based on the resulting model fit indices. A no coupling model is tested by constraining both cross lags to zero ($\gamma_{xy} = \gamma_{yx} = 0$). An equal dynamics bidirectional

model is tested by constraining all cross lags to be equal ($\gamma_{xy} = \gamma_{yx}$). Two unidirectional models are tested by allowing one cross-lag to be freely estimated while the opposing cross-lag is constrained to zero (γ_{xy} = 'free'; $\gamma_{yx} = 0$). Goodness of fit for four competing models were compared to the full dynamics model which was specified to allow auto-proportion (β) and cross lag (γ) coefficients to be freely estimated.

Multivariate adjusted models were also specified. Population level intercept and slope factors were regressed on baseline covariates to evaluate if background variables substantially altered dynamic interrelations. In a final step, the model was run on a sub-sample of participants who completed all four waves of assessment and had MMSE scores greater than 23. Follow-up analyses were conducted by further adjusting the leading indicator for time-varying covariates (medical conditions and visual acuity).

To illustrate the inter-domain coupling effects, model implied mean trajectories were graphed in a series of hypothetical scenarios where initial levels in each domain were either fixed at the sample mean intercept, fixed at one standard deviation above the mean intercept or fixed at one standard deviation below the mean intercept. The model implied mean trajectories were calculated with the following formulas:

$$X_{[t]} = 1 \times X_{slope} + (1 + \beta_x) \times X_{[t-1]} + \gamma_{yx} \times Y_{[t-1]}$$

$$Y_{[t]} = 1 \times Y_{slope} + (1 + \beta_y) \times Y_{[t-1]} + \gamma_{xy} \times X_{[t-1]}$$

Statistical software Mplus (Muthen & Muthen, 2007) was used for all analyses, and full information maximum likelihood (FIML) algorithms were used to account for missing data, which was assumed to be MAR (Little & Rubin, 2002).

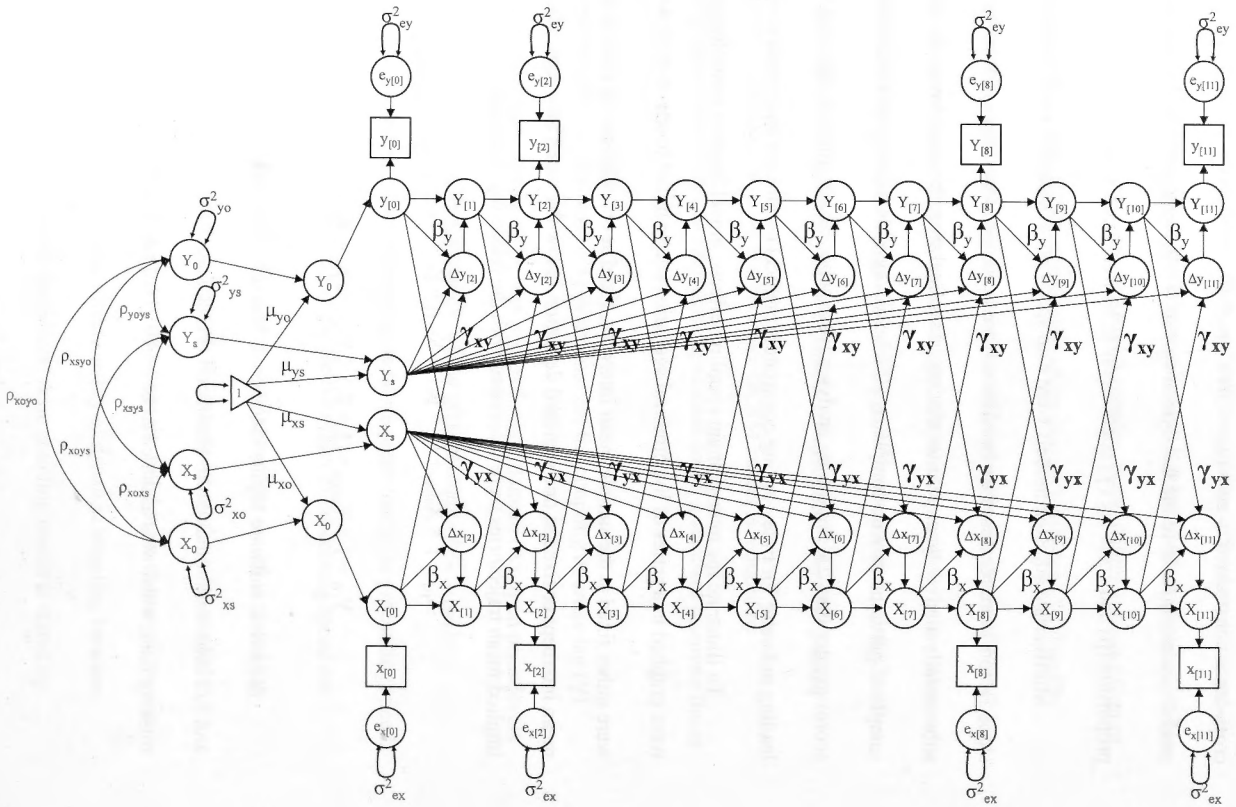

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Figure 8.2 Path diagram of an unadjusted Bivariate Dual Change Score Model used in the current study. Residuals were constrained to be equal within domain over time, but were not correlated across domains at each time point. The model can be adjusted for by regressing the intercept (X_0, Y_0) and slope (X_s, Y_s) factors on time invariant covariates, and residualising observed variables ($x_{[0]}$, $x_{[2]}$, $x_{[8]}$, $x_{[11]}$ and $y_{[0]}$, $y_{[2]}$, $y_{[8]}$, $y_{[11]}$) for time varying covariates.

Table 8.1 Sample characteristics for participants who completed both hearing and cognitive assessment at baseline.

Variable	n	*M*	(*SD*)
Hearing			
Baseline	1172	50.3	(9.8)
Year 2	919	49.4	(9.8)
Year 8	382	47.4	(9.6)
Year 11	278	45.9	(10.2)
Processing Speed			
Baseline	1172	50.2	(9.9)
Year 2	837	51.2	(10.1)
Year 8	342	51.1	(9.2)
Year 11	258	49.1	(9.8)
Covariates			
Age	1172	77.3	(6.2)
MMSE	1158	27.4	(2.6)
CESD	1138	7.7	(7.1)
Visual Acuity (logMAR)	945	0.3	(0.4)
Male	608	51.8%	
Noise exposure	282	24.1%	
Current Smoker	77	6.6%	
Hypertension	380	32.4%	
Stroke	28	2.4%	
Heart Attack	137	11.7%	
Diabetes	73	6.2%	

Note: Hearing thresholds and processing speed were assessed at baseline and at follow-up intervals of 2, 8 and 11 years. Both hearing thresholds and processing speed were transformed to T-scores (M=50, SD=10) standardized to the entire baseline sample.

Hearing: Pure Tone Average of 0.5, 1, 2, 4 kHz dB HL in the better ear.

Processing Speed: Digit Symbol Substitution Test.

MMSE: Mini Mental State Examination (Folstein et al., 1975b).

CESD: Center for Epidemiological Studies Depression Scale (Radloff, 1977).

8.3 Results

8.3.1 Comparison of competing models

Table 8.2 shows the goodness of fit indices for the unadjusted and adjusted models tested by the DCSM. All models had acceptable levels of fit (CFI>0.95, RMSEA<.05) indicating they were a good match to the data. For the unadjusted models, optimal fit was obtained when cross-lag (γ) paths between processing speed latent difference scores (ΔDSS_t) and prior hearing levels (PTA_{t-1}) were constrained to zero (CFI = .988). Both BIC and Chi-square differences revealed that this was the only model that did not show reduced goodness of fit relative to the full dynamics model ($\Delta\chi^2$=0.1, Δdf=1, p = .75). From this we can infer a unidirectional association whereby hearing thresholds were leading indicators of change of processing speed. This pattern remained after adjusting for covariates. The parameter estimates from the adjusted full dynamics model are presented in Table 8.3.

8.3.2 Differential Magnitude of Inter-domain Coupling

The DCSM consists of a complex train of between and within domain regression paths and is therefore difficult to directly interpret individual coefficients independently of all other estimates. For this reason graphical examples of six hypothetical scenarios that demonstrate the differential magnitude of the coupling parameters and their directional effects over time are presented in Figure 8.3. Each scenario shows how the cross-lag parameters (γ) predict the estimated mean trajectories of a coupled variable. Figures were produced using estimates from the unadjusted full coupling model (μ_{DSS_slope} = -0.98, *SE* = 1.71; μ_{PTA_slope} = -5.09, *SE* = 1.35; β_{DSS} = -0.33, *SE* = 0.06; β_{PTA} = 0.66, *SE* = 0.45; $\gamma_{DSS\rightarrow\Delta PTA}$ = 0.03, *SE* = 0.05; $\gamma_{PTA\rightarrow\Delta DSS}$ = 0.35, *SE* = 0.05) and the formulas:

$$X_{[t]} = 1 \times X_{slope} + (1 + \beta_x) \times X_{[t-1]} + \gamma_{yx} \times Y_{[t-1]}$$

$$Y_{[t]} = 1 \times Y_{slope} + (1 + \beta_y) \times Y_{[t-1]} + \gamma_{xy} \times X_{[t-1]}$$

The upper panel of Figure 8.2 depicts the model implied means of change in PTA and DSS over 11 years for participants with mean intercepts in both domains (M_{PTA} = 50.18; M_{DSS} = 50.24). From this, it is evident that on average, both hearing and processing speed declined over the 11 year period. The right side panel of Figure 8.3 depicts 4 hypothetical scenarios where the initial levels of either PTA or DSS are increased or decreased half a standard deviation. The purpose of this is to illustrate how varying the initial levels in one domain can alter the estimated mean trajectory in the coupled domain. Altering the initial levels of PTA had a noticeable impact on estimated DSS trajectories whereas altering the initial DSS levels had marginal impact on estimated PTA trajectories.

Regrettably, attempts to include time varying covariates in the specified model resulted in an MPLUS warning of un-trustworthy standard errors due to a non-positive definite first-order derivative product matrix. Moreover, graphing of the model implied means indicated improbable trajectories that oscillated around the mean, suggesting an unstable model and unreliable estimates. Thus, even though fit indices indicated that hearing remained a leading indicator of change in processing speed, these particular analyses were not interpreted.

As a final sensitivity analysis, the full multivariate-adjusted model was fitted to a sub-sample of participants with complete data at all waves who had baseline MMSE scores greater than 23 (n=119). The results of these analyses were more reliable than the

multivariate adjusted model fitted to data from the full sample and again indicated that hearing thresholds were leading indicators of decline in processing speed (Figure 8.4).

Table 8.2 Goodness of fit indices for unadjusted zero-order bivariate DCSMs (top) and adjusted bivariate DCSMs (bottom) of hearing thresholds and processing speed over 11 years.

Model	χ^2	(df)	$\Delta\chi^2$	(Δdf)	BIC	CFI	RMSEA
			Unadjusted models				
No coupling							
No dynamics	114.3	(26)	30.8*	(2)	36579	.974	.054
Unidirectional coupling							
Speed→ΔHearing	112.0	(25)	28.6*	(1)	36584	.974	.054
Hearing→ΔSpeed	83.7	(25)	0.2	(1)	36555	.982	.045
Bidirectional coupling							
Equal dynamics	99.0	(25)	15.5*	(1)	36570	.978	.050
Full dynamics	83.4	(24)	-	-	36562	.982	.046
	Adjusted for age, sex, MMSE, CESD, and noise exposure						
No coupling							
No dynamics	135.0	(46)	41.2*	(2)	59659	.978	.041
Unidirectional coupling							
Speed→ΔHearing	131.4	(45)	37.6*	(1)	59662	.979	.040
Hearing→ΔSpeed	93.6	(45)	-0.3	(1)	59625	.988	.030
Bidirectional coupling							
Equal dynamics	145.9	(45)	52.0*	(1)	59677	.975	.044
Full dynamics	93.9	(44)	-	-	59632	.988	.031

Note: n = 1172; **Hearing**: Pure Tone Average of 0.5, 1, 2, 4 kHz dB HL in the better ear. **Speed:** Digit Symbol Substitution Test. BIC = Bayesian Information Criteria; CFI = Comparative Fit Index; RMSEA = Root mean Square Error of Association. PTA = Pure Tone Average of 0.5, 1, 2, 4 kHz dB HL in the better ear. Significance values refer to loss in χ^2 value relative to the full dynamics model.

* $p < .01$

Table 8.3 Parameter estimates from adjusted bivariate Dual Change Score Model with full dynamics between processing speed and pure tone average of hearing thresholds in the better ear and (n = 1072).

	Hearing Thresholds		Processing Speed		Cross-domain effects	
	Est.	**SE**	**Est.**	**SE**	**Est.**	**SE**
Fixed Effects						
Intercept mean (μ_o)	51.45**	0.45	50.76**	0.45		
Slope mean (μ_s)	-4.81**	1.23	-2.61	1.69		
Proportion (β)	0.10*	0.05	-0.34**	0.06		
Random Effects						
Intercept variance (σ^2_o)	62.58**	2.96	51.50**	3.12		
Slope variance (σ^2_s)	0.76	0.59	13.77**	4.05		
Error variance (σ^2_e)	12.96**	0.56	20.62**	1.16		
Covariance within domains						
Intercept ↔ Slope	-6.46*	2.63	10.95**	2.43		
Dynamics						
$\gamma_{PTA \rightarrow \Delta DSS}$					0.39**	0.06
$\gamma_{DSS \rightarrow \Delta PTA}$					-0.02	0.05
Covariance between domains						
$PTA_{intercept} \leftrightarrow DSS_{intercept}$					4.99*	2.12
$PTA_{slope} \leftrightarrow DSS_{slope}$					2.47	1.72
$PTA_{intercept} \leftrightarrow DSS_{slope}$					-21.73**	3.48
$PTA_{slope} \leftrightarrow DSS_{intercept}$					0.41	1.87

Note: Hearing thresholds and processing speed were transformed to a t-score metric using the baseline sample as the reference. All estimates are unstandardized and residualized for age (centered at 75 years), sex, cognitive impairment (MMSE), depressive symptoms (CES-D) and self-reported occupational noise exposure. Model fit: χ^2 (44) = 93.8; CFI = .988; RMSEA = .031.

* $p < .05$; ** $p < .01$

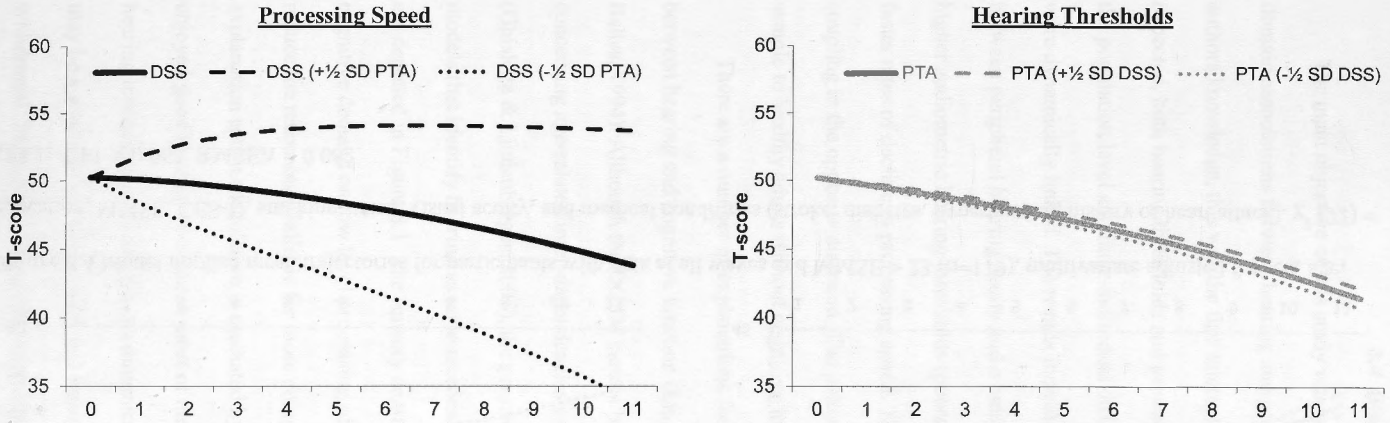


Figure 8.3 Model implied means trajectories for Digit Symbol Substitution (DSS) scores (black lines upper panel) and Pure-Tone Average (PTA) of hearing thresholds in the better ear (grey lines, lower panel) estimated by the unadjusted Full Dynamics DCSM (n=1072). Varying the initial level for processing speed has minimal effect on the estimated mean rate of change in hearing thresholds. In contrast, varying the initial level of hearing will noticeably alter the estimated mean rate of change in processing speed. Higher levels of hearing were associated with more gradual rates of declines in processing speed (black line, long dash) whereas lower levels of hearing were associated with faster rates of declines in processing speed (black line, short dash). All variables are transformed to a t-score metric (M=50, SD=10) using the baseline sample as reference and y-axis scales were orientated so a negative gradient indicates a decline in performance.

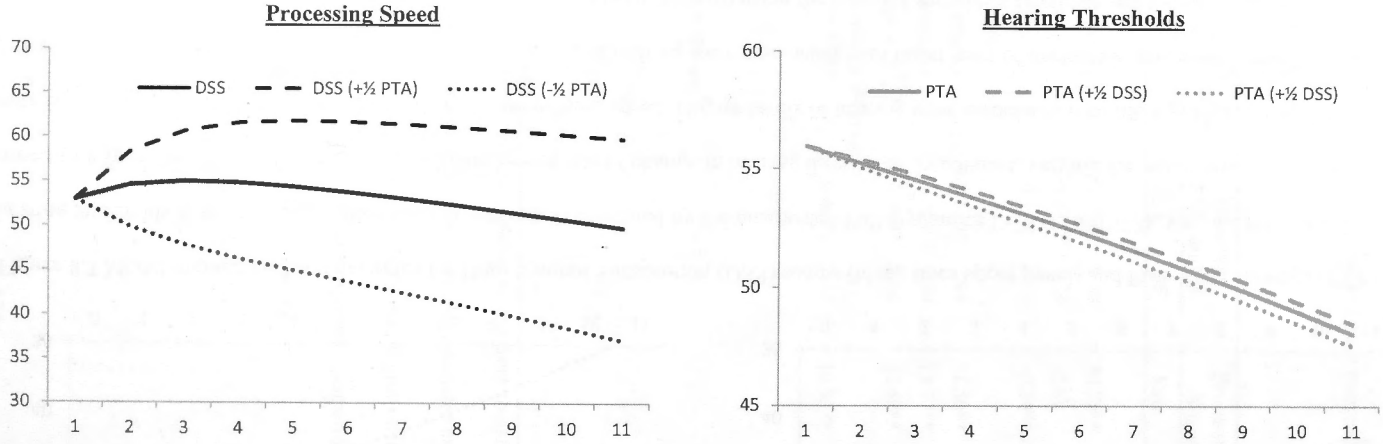


Figure 8.4 Model implied mean trajectories for participants with data at all waves and MMSE > 23 (n=119), multivariate adjusted for sex, age, education, MMSE, CES-D, smoking status, visual acuity, and medical conditions (stroke, diabetes, hypertension, history of heart attack). χ^2 (72) = 105.1, CFI = 0.963, RMSEA = 0.062.

8.4 Discussion

The main objective of this study was to investigate long term dynamic inter-domain associations between hearing and processing speed during late life. To the authors knowledge, this was the first attempt to apply DCSMs to audiometric data. As expected, both hearing thresholds and processing speed underwent decline over time at the population level and inter-individual differences in these longitudinal trajectories were dynamically linked. The results indicate that there is a unidirectional coupling between peripheral hearing acuity and a measure of cognitive ageing. Specifically, higher audiometric hearing thresholds (poorer hearing levels) preceded and predicted faster rates of decline in processing speed. There was no evidence of inter-domain coupling in the opposite direction. The pattern of findings remained after restricting the sample to healthy adults without cognitive impairment who participated at all 4 waves.

There are a number of explanations for the directional cross domain coupling between hearing and cognitive function (Lin, Ferrucci, et al., 2011; Lindenberger & Baltes, 1994). Although the DCSM cannot be used to reject competing hypotheses concerning age-related inter-dependencies between sensory and cognitive function (Ghisletta & Lindenberger, 2005), the present findings do lend stronger support to models that identify hearing as an antecedent to cognitive decline. Two such hypotheses are depicted in Figure 8.1. The sensory degradation hypothesis maintains that increased cognitive demand on low level processing of auditory stimuli could increase fatigue and reduce the resources available for more complex cognitive processes. An alternate explanation is that the relation is mediated by a social mechanism. Adults who have enjoyed good hearing throughout most of their life may find it challenging to adjust to hearing loss acquired in late-life. Communication difficulties arising from hearing loss may be a source of embarrassment and frustration, which could be a catalyst for social withdrawal. Thus, sensory impairment may limit opportunities to engage in stimulating

activities that are purported to be important for building cognitive reserve. Further, this social isolation means hearing loss may indirectly give rise to depression which has been identified as a risk factor for decline in processing speed using dynamic bivariate DCSMs (Bielak et al., 2011).

A common cause model cannot be directly tested by the DCSM. It may be that brain ageing affects peripheral hearing before it affects processing speed. As the results remained unchanged when restricted to a sub-sample of healthy participants who did not have cognitive impairment, and given dementia neuropathology does not affect the cochlear or inner ear, it could be argued that this evidence in favour of a common cause is weak. However, adjusting the model for health covariates that may be proxies for a common cause is an imperfect and unsatisfying solution to testing this hypothesis. Thus degradation of the central nervous system, or brain ageing neuropathology such as those associated with Alzheimer's Disease (Anstey, 2008b), remain further possible explanations that cannot be dismissed.

These findings are also consistent with biomarker theories of ageing (Anstey, 1999; Anstey & Smith, 1999a). If age is at best a proxy for mechanistic changes that drive cognitive decline, then perhaps other ageing processes better placed to provide a more illuminating account of cognitive ageing phenomena (MacDonald et al., 2011; Sliwinski & Mogle, 2008). Peripheral hearing level may be a good biomarker of a person's true functional age, alongside other functional biomarkers such as visual acuity, grip-strength, lung function and blood pressure. Because sensory variables are inextricably linked to cognition via perceptual processing, the status of sensory acuity as a biomarker of cognitive ageing has been elevated (Anstey, 2008b). When compared to vision, hearing has been considered less useful as a sensory biomarker due to the strong link between hearing loss and environmental exposures like noise and solvents.

This raises the question, to what extent are these findings specific to hearing or do they apply to sensory abilities in general.

The reported uni-directional temporal ordering of hearing and processing speed contrasts with the exploratory findings by Ghisletta and Lindenberger (2005), who also used DCSMs to test time-ordered inter associations between vision and cognition. They reported bi-directional coupling between near vision and processing speed. On the basis of their findings the Ghisletta and Lindenberger argued a cascade hypothesis was not strongly supported. Further, a number of studies that identified links between visual acuity and MMSE (Lin et al., 2004; Reyes-Ortiz et al., 2005), processing speed or memory (Anstey et al., 2003a; Gussekloo, de Craen, Oduber, van Boxtel, & Westendorp, 2005) failed to find similar effects for hearing. The apparent discrepancies between these previous studies and the current findings can be reconciled. Those studies that did not find links between hearing and cognition were either cross-sectional, had shorter follow-up periods, or operationalized sensory loss as binary rather than continuous variables, or applied different analytic models to the DCSM so were testing a different set of research questions to those presented here. Care must also be taken not to draw too many parallels between vision and hearing, as dementia neuropathology and brain ageing can directly affect retinal function (Guo, Duggan, & Cordeiro, 2010; Kuljis, 2001). Moreover, it is highly likely that the visual presentation of some cognitive tests could result in inflated or even spurious associations. Task impurity artefacts could explain why some researchers have reported associations between vision and cognition but not hearing. This is therefore a strength of the current study as the Digit Symbol Substitution Test shares few task parameters with pure-tone audiometry.

8.4.1 Limitations

Limitations of the DCSM have been discussed at length (e.g. Gerstorf et al., 2007) and a number of methodological and substantive caveats to the present findings must be recognised. Firstly, these findings do not imply causation, as only associations between prior levels of hearing and declines in processing speed were demonstrated. Secondly, all couplings within and between domains were estimated at the population level as fixed effects so it is not possible to draw direct inferences at the level of the individual without making the unlikely assumption of equivalence of inter-individual and intra-individual variation. In other words, as with other standard analytic techniques such as regression, the DCSM is subject to Ergodicity (Molenaar, 2008; Molenaar & Campbell, 2009). Further, many of the model parameter estimates were invariant across time, for example both the cross lag and auto-proportion weights were equal at all time points. Although the DCSM is a complex statistical model, it represents a simple system.

Unfortunately there have been no published simulations on the power of DCSMs. It has been demonstrated that multivariate growth curve models are often underpowered, particularly when there are small number of repeated observations (Hertzog, von Oertzen, Ghisletta, & Lindenberger, 2008). Discussion with other users of the DCSM indicate that variables with larger auto-proportions (β weights) have greater power to exert cross lag effects, and larger error variance reduce the chances of receiving a cross lag effect. This is encouraging as processing speed had larger auto proportions and larger error variance yet was shown to be reliably predicted by lagged hearing levels while wielding no reliable effect on hearing thresholds.

8.4.2 Conclusions and Considerations for Future Research

In summary, the present findings indicate that hearing thresholds in the better ear are leading indicators of decline in processing speed, extending previous observations which have shown peripheral hearing loss to be cross-sectionally associated with

processing speed and also predictive of future dementia diagnoses. The strengths of this study are the large sample size with 11-years of follow up data and the visually administered test of processing speed. It is unlikely that the results are confounded by participants with poor hearing experiencing difficulty in completing the DSS. Aside from the need for replication, the implications of these results point to a number of promising avenues where future investigations may focus their efforts. It would be insightful if future research extended these findings and investigated the long term coupling relations between central auditory functioning and cognition. Further, if hearing loss impacts on cognition via over-taxing and monopolizing available resources allocation for lower level tasks, or if hearing loss reduces opportunities to engage of activities purported to build and maintain cognitive reserve, then it might be expected that early utilisation of hearing aids may attenuate rates of decline in cognition among adults with acquired age-related hearing loss.

CHAPTER 9: Hearing Aid Use and Cognitive Decline

Synopsis

The mechanism that underpins the inter-association between hearing and cognitive function is unclear. One approach to evaluating sensory degradation is to employ a quasi-experimental design to test if use of sensory aids predicts slower rates cognitive decline. It has been suggested that hearing aid use may delay or slow the progression of cognitive decline among older adults. In this Chapter, linear and generalized mixed models were used to evaluate whether regular hearing aid use predicted higher levels of and slower rates of change cognitive function. It was found that although hearing aid use was associated with faster levels of processing speed and improved performance on the MMSE, rates of change were not reliably different after the commencement of hearing aid use. There were also both between-person and within person effects of hearing thresholds on processing speed. A number of explanations are offered, including the suggestion that aid use may help older adults maintain and build their cognitive reserve leaving them better equipped to cope with cognitive load attributable to listening difficulties. The possibility of reciprocal causation is also explored. Hearing impaired adults with good levels of cognitive function may experience fewer difficulties acclimatizing to hearing aids and may be better placed to take full advantage of their benefits.

9.1 Background

The previous chapter demonstrated a dynamic link between hearing and fluid cognitive abilities, revealing that poor levels of hearing are associated with future declines in processing speed. However, the extent to which these findings could be explained by either biological or social factors could not be determined. One approach to evaluate the role of sensory degradation as a determinant of cognitive decline is to test if use of sensory aids attenuate rates of cognitive decline among adults with low levels of sensory function (Valentijn, Van Boxtel, et al., 2005). Hearing aid use has been proposed to be protective against age-related cognitive decline (Arlinger et al., 2009; Cacciatore et al., 1999; Choi, Shim, Lee, Yoon, & Joo, 2011; Pichora-Fuller & Singh, 2006). Such a proposition is considered of major importance to both theorists and practitioners within cognitive-hearing science, for example, Lin (2012, page 1148) has described the hypothesis that hearing treatment could delay cognitive impairment to be the 'most salient question at hand' for hearing clinicians.

There are a number of explanations that justify why we might expect hearing aid use to predict better cognitive function. Difficulty understanding speech is taxing on cognitive resources. Hearing aids may reduce the cognitive load and excess demands made by auditory perception and allow these cognitive resources to be allocated to other higher level processes. Adults with impaired hearing who use hearing aids have been shown to have improved quality of life (Appollonio, Carabellese, Frattola, & Trabucchi, 1996) and increased social engagement (Davis et al., 2007), allowing continued participation in activities that maintain, or perhaps even build, their cognitive reserve. Both these explanations draw upon cascade models and sensory degradation hypotheses (Figure 9.1). Alternatively, hearing aid use could directly enhance cognitive reserves as regular use of a hearing aid can be construed as a type of cognitive training in and of itself. It can take up to three months to properly acclimatize to hearing aids (Weinstein,

2000). Learning to use and become comfortable with a new listening environment requires the processing of new and complex auditory stimuli, which could promote brain plasticity. On the other hand, good cognitive function is a determinant of successful acclimatization as it facilitates faster and easier adaptation to a new listening environment (Pichora-Fuller, 2009; Pichora-Fuller & Singh, 2006). Along with working memory and attentional control, processing speed is one cognitive function considered to be important for listening, and has been argued to play a crucial role in acclimatizing to hearing aids (Beck, 2011; Cohen, 1987). Thus, there could potentially be multifaceted dynamic interactions between hearing aid use and cognitive decline.

The benefits of hearing aid use on quality of life are uncontroversial and this is where most research has been focused (Lin, 2012), but the evidence that these benefits extend to the cognitive domain is unclear. Three studies have reported that adults fitted with hearing aids showed improved levels of cognitive functioning (working memory and verbal learning), within 6 months when compared to unaided controls (Choi et al., 2011; Lehrl, Funk, & Seifert, 2005; Mulrow et al., 1990). In contrast, hearing aids have not been found to predict improved performance on other neurocognitive tests, including measures of processing speed and verbal fluency (Tesch-Romer, 1997; van Hooren et al., 2005). Non-verbal cognitive assessments were used in studies reporting both null and positive results. All these studies employed randomised control designs in clinical settings, and can be expected to have closely monitored hearing aid fitting procedures and participant usage. However, they are of modest sample size of a few hundred participants at most, and are restricted to two measurement occasions over short a follow-up period, which reduces their capacity to effectively model differences in rates of change.

There have been few investigations of the effects of hearing aids on cognitive function in population based studies. Longitudinal analyses of three waves of data from

the Maastricht Aging Study did not find hearing aid use to be related to baseline cognitive function or cognitive decline over 6 years. Although power constraints may again have obscured any association as this sample had 44 participants reporting use of a hearing aid at baseline and only a further 7 participants were fitted with a hearing aid during the course of the study (Valentijn, Van Boxtel, et al., 2005). This chapter takes a similar approach to Valentijn (2005) by investigating long-term links between hearing aid use and cognitive decline by analysing longitudinal data from BMES and ALSA.

There have been two reports on hearing aid use within Australia that draw on ALSA or BMES. Hearing aid use has recently been reported for ALSA (Sanchez et al., 2011), with higher levels of hearing aid use in wave 9 than at baseline. However, hearing aid use in ALSA has not been investigated in relation to age-related cognitive decline. A study of BMES investigating predictors of hearing aid ownership did not find an association between cognitive impairment and incidence of hearing use (Gopinath et al., 2011), though with only 26 participants identified with cognitive impairment (MMSE < 24) these findings were underpowered.

Another aspect of hearing aid use that may be pertinent to the aims of this study is the frequency of usage. Difficulty and dissatisfaction with hearing aids increases the risk of irregular usage (Bertoli et al., 2009). The persistent use of hearing aids may therefore be an important factor to consider when exploring links between auditory functioning, hearing aid use and cognition. It might be expected that stronger benefits are found among adults who use hearing aids on a more frequent basis.

The aims of this chapter are to test the associations between hearing aid use and cognitive decline after adjusting for degree of hearing loss. A naturalistic quasi-experimental design is employed, with hearing aid use being the treatment condition and cognition the outcome. It is predicted that adults reporting use of hearing aids will have higher levels and more gradual rates of change in cognition after the

commencement of hearing aid use. It is also expected that more frequent and persistent hearing aid use will confer greater benefits. The methods and results are presented as two studies. In the first study four waves of pooled ALSA and BMES data are analysed, with MMSE score as the outcome. In the second study five waves of ALSA data are analysed with processing speed as the outcome.

9.2 Study 1 Methods

9.2.1 Study1 Participants and Measures

This study draws on the same sample as presented in Chapter 7. Briefly, data from participants in the pooled ALSA and BMES sample who completed cognitive and hearing assessment were included in the analyses. Participants were tested on up to 4 occasions over 11 years. The outcome measure was number of errors on the MMSE. The main independent variable of interest was self-reported hearing aid use at each wave. Time invariant covariates were age at baseline, sex, study and highest qualification (secondary school only, post-secondary non-tertiary, tertiary). Time varying covariates included PTA in the better ear, impaired visual acuity (< 0.3 logMAR) and self-reported diabetes, stroke, and hypertension.

9.2.2 Study 1 Analyses

The main purpose of these analyses was to investigate if rate of change in number of MMSE errors decreased after participants began reporting hearing aid use. To address this aim, piecewise mixed models with fixed and random effects were used to test for continuous and discontinuous change following the procedure described by Singer and Willet (2003). There were three key independent variables in this model. Time (years) was included to model rates of change in hearing thresholds. A time varying indicator of hearing aid use was included to reflect differences in intercepts (cross sectional elevation) between hearing users and non-hearing aid users. It was possible for participants to quit hearing aid use. A second time variable reflecting the years a respondent had been using a hearing aid was included to model a discontinuity in rate of change among hearing aid users. All three of these variables were included as both fixed and random effects. An example of how the dataset was formatted to facilitate these analyses is provided in Table 9.1.

Model-building followed a hierarchical procedure whereby a number of competing and nested models of increasing complexity were tested. The equations for these models are presented in Table 9.2. Initially, linear mixed model (LMM) unconditional means (UM) and unconditional growth (UG) models were fitted to MMSE data. Then in LMM 1, fixed effects for baseline age and time-varying PTA were modelled. This became the baseline reference model for evaluating the effects of hearing aid use. To evaluate discontinuities in intercepts associated with hearing aid use, LMM 2 incorporated fixed and random effects for time-varying hearing aid use (Aid). To evaluate discontinuities in trajectories after hearing aid use, LMM 3 incorporated fixed and random effects for time using a hearing aid (Post-Aid Time). These three steps were repeated with the inclusion of socio-demographics (sex and education) and health covariates (medical conditions and visual impairment) in LMMs 4-6. At each step BIC, AIC and -2ll values were evaluated to assess if the inclusion of additional model parameters significantly improved model fit.

Due to the distributional properties of the MMSE, such as strong ceiling/floor effects, there are concerns that it may be inappropriate to model MMSE data using linear mixed models and that this cannot be addressed by transformations. Failure to properly account for this may result in spurious estimates and sub-optimal model fit (Proust-Lima et al., 2011). Although it has been argued that this concern is overstated, with simulation studies showing that linear regression can produce valid results for non-normal outcomes in epidemiological data with large sample sizes (Lumley, Diehr, Emerson, & Chen, 2002). Thus, both linear mixed models and generalized latent mixed models were tested and their results compared. Visual inspection of normality plots (histograms, normal probability curve, and scatter plot of standardized residuals) for the residuals estimated from the linear mixed model were also made to assess violations of normality. All analyses were conducted using Stata IC version 10 (StataCorp, 2007).

Table 9.1 Example of data format for modelling discontinuity in level and change.

ID	MMSE (errors)	Time (years)	Post-Aid Time (years)	Aid	PTA (dB)	Age at baseline
1	1	0.0	0.0	0	17.5	78
1	1	2.0	0.0	0	21.3	78
1	0	8.0	0.0	0	32.5	78
2	0	0.0	0.0	0	26.3	72
2	2	2.1	0.0	0	25.0	72
2	1	7.9	0.0	1	31.3	72
2	6	11.0	3.1	1	35.0	72
3	0	0.0	0.0	0	40.0	84
3	3	2.0	0.0	1	42.5	84
3	1	7.9	5.9	1	48.8	84
3	5	11.0	9.0	1	47.5	84

Table 9.2 Level 1 (individual level) and Level 2 (population average level) equations tested for linear mixed models

Model	Level 1	Level 2
LMM 1	$MMSE_{ij} = \pi_{0i} + \pi_{1i}Time_{ij} + \pi_{2i}PTA_{ij} + \varepsilon_{ij}$	$\pi_{0i} = \gamma_{00} + \gamma_{01}Age_i + \zeta_{0i}$ $\pi_{1i} = \gamma_{10} + \gamma_{11}Age_i + \zeta_{1i}$ $\pi_{2i} = \gamma_{20}$
LMM 2	$MMSE_{ij} = \pi_{0i} + \pi_{1i}Time_{ij} + \pi_{2i}PTA_{ij} + \pi_{3i}Aid_{ij} + \varepsilon_{ij}$	$\pi_{0i} = \gamma_{00} + \gamma_{01}Age_i + \zeta_{0i}$ $\pi_{1i} = \gamma_{10} + \gamma_{11}Age_i + \zeta_{1i}$ $\pi_{2i} = \gamma_{20}$ $\pi_{3i} = \gamma_{30} + \zeta_{3i}$
LMM 3	$MMSE_{ij} = \pi_{0i} + \pi_{1i}Time_{ij} + \pi_{2i}PTA_{ij} + \pi_{3i}Aid_{ij} + \pi_{4i}Time\_PostAid_{ij} + \varepsilon_{ij}$	$\pi_{0i} = \gamma_{00} + \gamma_{01}Age_i + \zeta_{0i}$ $\pi_{1i} = \gamma_{10} + \gamma_{11}Age_i + \zeta_{1i}$ $\pi_{2i} = \gamma_{20}$ $\pi_{3i} = \gamma_{30} + \zeta_{3i}$ $\pi_{4i} = \gamma_{40} + \zeta_{4i}$
LMM 4	LMM 1 + time-varying medical conditions (stroke, hypertension, diabetes), visual impairment, sex and education	
LMM 5	LMM 2 + time-varying medical conditions (stroke, hypertension, diabetes), visual impairment, sex and education	
LMM 6	LMM 3 + time-varying medical conditions (stroke, hypertension, diabetes), visual impairment, sex and education	
GLZMM 1	$\ln(MMSE_{ij}) = \pi_{0i} + \pi_{1i}Time_{ij} + \pi_{2i}PTA_{ij} + \varepsilon_{ij}$	$\pi_{0i} = \gamma_{00} + \gamma_{01}Age_i + \zeta_{0i}$ $\pi_{1i} = \gamma_{10} + \gamma_{11}Age_i + \zeta_{1i}$ $\pi_{2i} = \gamma_{20}$
GLZMM 2	$\ln(MMSE_{ij}) = \pi_{0i} + \pi_{1i}Time_{ij} + \pi_{2i}PTA_{ij} + \pi_{3i}Aid_{ij} + \varepsilon_{ij}$	$\pi_{0i} = \gamma_{00} + \gamma_{01}Age_i + \zeta_{0i}$ $\pi_{1i} = \gamma_{10} + \gamma_{11}Age_i + \zeta_{1i}$ $\pi_{2i} = \gamma_{20}$ $\pi_{3i} = \gamma_{30}$
GLZMM 3	$\ln(MMSE_{ij}) = \pi_{0i} + \pi_{1i}Time_{ij} + \pi_{2i}PTA_{ij} + \pi_{3i}Aid_{ij} + \pi_{4i}Time\_PostAid_{ij} + \varepsilon_{ij}$	$\pi_{0i} = \gamma_{00} + \gamma_{01}Age_i + \zeta_{0i}$ $\pi_{1i} = \gamma_{10} + \gamma_{11}Age_i + \zeta_{1i}$ $\pi_{2i} = \gamma_{20}$ $\pi_{3i} = \gamma_{30}$ $\pi_{4i} = \gamma_{40}$

GLZMM 4	$\ln(MMSE_{ij}) = \pi_{0i} + \pi_{1i}Time_{ij} + \pi_{2i}PTA_{ij} + \pi_{3i}Aid_{ij} + \pi_{4i}Time\_PostAid_{ij} + \varepsilon_{ij}$	$\pi_{0i} = \gamma_{00} + \gamma_{01}Age_i + \zeta_{0i}$ $\pi_{1i} = \gamma_{10} + \gamma_{11}Age_i + \zeta_{1i}$ $\pi_{2i} = \gamma_{20}$ $\pi_{3i} = \gamma_{30} + \zeta_{3i}$ $\pi_{4i} = \gamma_{40} + \zeta_{4i}$

Note: Although a greater number of models were tested, only 8 are presented to save on space here as they best illustrate the model building procedure.

Time-varying medical conditions were not specified to have random effects.

LMM: Linear Mixed Model

GLZMM: Generalized Mixed Model

$\boldsymbol{MMSE_{ij}}$: MMSE errors for person *i* at time *j*

$\boldsymbol{\pi_{xi}}$: person *i*'s predicted mean value for time-varying covariate *x*

$\boldsymbol{\varepsilon}$: Level 1 residual

$\boldsymbol{\gamma}$: Population average fixed effects

$\boldsymbol{\zeta}$: Level 2 residuals, indicates that random effects are specified for a given time-varying covariate

Table 9.3 Goodness of fit statistics for eleven mixed models for change in MMSE errors (n = 3,412).

Model	BIC	AIC	-2ll	df	$\Delta\chi^2$	(Δdf)	*P*	σ_ε^2	Pseudo R^2	ΔPsuedo R^2	Comparison Model
Unconditioned linear models											
LMM UM	28769	28749	28743	3		-		5.17		-	
LMM UG	28412	28372	28360	6	382.5	(3)	<.001	3.89	24.9%	24.9%	Model UM
Age and PTA adjusted linear models											
LMM 1	27656	27582	27560	11		-		3.89	24.7%	-	
LMM 2	27619	27519	27489	15	71.5	(4)	<.001	3.78	26.8%	2.1%	Model 1
LMM 3	27651	27517	27477	20	12.2	(5)	.032	3.75	27.5%	0.7%	Model 2
Full adjusted linear models											
LMM 4	27519	27405	27371	17		-		3.90	24.5%	-	
LMM 5	27489	27348	27306	21	64.3	(4)	<.001	3.78	26.8%	2.3%	Model 4
LMM 6	27520	27345	27293	26	13.1	(5)	.023	3.76	27.3%	0.5%	Model 5
Age and PTA adjusted generalized models											
GLZMM 1	23219	23145	23123	11							
GLZMM 2	23223	23142	23118	12	5	(1)	.025				GLZMM 1
GLZMM 3	23219	23125	23098	14	20	(2)	.001				GLZMM 2
GLZMM 4	23258	23130	23093	19	5	(5)	.416				GLZMM 3

σ_ε^2: level 1 residual. LMM: Linear Mixed Model; GLZMM: Generalized Mixed Model

Note: Full adjusted models include all fixed effects for sex, education, medical conditions, and visual impairment.

Of the Linear Mixed Models, LMM 5 has the smallest BIC value, but LMM 6 has the smallest AIC and -2ll values. Test of chi-square difference indicates that LMM 6 has closer fit to the data compared to LMM 5. Overall, GLZMM 3 has the optimal and most parsimonious model fit.

Table 9.4 Estimates for trajectories of MMSE errors with discontinuities in elevation and slope as a function of hearing aid use (n = 3412).

		Age and PTA Adjusted (LMM 3)			Multivariate Adjusted (LMM 6)			Age and PTA Adjusted (GLZMM 4)		
		Est.	*p*	95% CI	Est.	*p*	95% CI	Log(IRR)	*p*	95% CI
Fixed Effects										
Intercept	γ_{00}	1.97	<.001	(1.87, 2.07)	2.39	<.001	(2.24, 2.55)	0.37	<.001	(0.31, 0.42)
Slope (years)	γ_{10}	0.18	<.001	(0.16, 0.20)	0.16	<.001	(0.14, 0.19)	0.08	<.001	(0.06, 0.09)
Aid	γ_{20}	-0.59	<.001	(-0.84,-0.35)	-0.51	<.001	(-0.76,-0.27)	-0.19	<.001	(-0.30,-0.09)
Post-Aid Slope	γ_{30}	-0.08	.027	(-0.16,-0.01)	-0.08	.041	(-0.15, -0.0)	-0.02	.083	(-0.05, 0.00)
Random Effects										
Intercept	σ_0^2	1.72		(1.40, 2.12)	1.58		(1.27, 1.98)	0.71		(0.63, 0.78)
Slope	σ_1^2	0.01		(0.01, 0.03)	0.01		(0.00, 0.03)	0.01		(0.01, 0.01)
Aid	σ_2^2	3.18		(1.87, 5.39)	3.14		(1.83, 5.37)	0.45		(0.00, 0.04)
Post-Aid Slope	σ_3^2	0.09		(0.02, 0.36)	0.10		(0.03, 0.32)	0.01		(0.26, 0.81)
Residual	σ_ϵ^2	3.75		(3.48, 4.04)	3.76		(3.49, 4.05)	-		-
Model Fit										
BIC		27651			27520			23092		
-2ll (df)		27477	(20)		27345	(26)		23258	(19)	

Note: All models are adjusted for age and PTA. The multivariate adjusted model also includes time-varying predictors of hypertension, stroke, diabetes, visual impairment, as well as time-invariant predictors of sex and education. Effects for these covariates are not shown due to space constraints and because they are not part of the focus of this study. Covariance components between random effects were estimated, but are also not

shown due to space constraints. The only significant covariance component was between Intercept and Aid (Model 3: σ_{02}=-0.92, SE=0.40; Model 6: σ_{02}=-0.85, SE=0.41). There was also significant covariance between Intercept and Aid in the GLZ model (σ_{02}=-0.24, SE = 0.06).

9.3 Study 1 Results

9.3.1 Hearing aid use over time

At baseline there were 389 (11.1%) participants who completed MMSE and hearing assessment reporting the use of a hearings aid. Only 8.4% of adults with Mild Hearing Loss and 44.7% of adults of moderate Hearing Loss reported using a hearing aid. At wave 2, incidence of hearing aid use was reported by a further 180 participants (7.5%) who were not using aids at baseline. The incidence of hearing aid use at waves three and four, was 56 participants (13.5%) and 40 participants (14.3%) respectively.

9.3.2 LMM Goodness of Fit

Model fit statistics for the competing models are shown in Table 9.3, there was some ambiguity among the goodness of fit indices. Whereas LMM 5 had the smallest BIC value, LMM 6 had the smallest AIC and -2ll values. Further, although the inclusion of fixed and random effects for post-aid slope in LMM 6 only explained a further 0.5% of the level-1 residual variance, a test of chi-square difference indicated that their inclusion made a significant improvement to model fit relative to LMM 5. It was therefore concluded that there were discontinuities in both intercepts and slopes after participants began reporting use of hearing aids.

9.3.3 LMM Parameter Estimates

The estimates for LMM 3 and LMM 6 are shown in Table 9.4. Both models indicate that use of hearing aid was associated with fewer MMSE errors and a slower rate of increase in MMSE errors over time. A graphical illustration of this is presented in Figure 9.1 which depicts trajectories for three hypothetical individuals who differ only in their patterns of hearing aid use over time. Each hypothetical individual represents a 75 year old woman with a mild degree of hearing impairment at baseline and average increase in hearing thresholds each year, but otherwise good health. There

was a mean difference of only 0.51 MMSE errors between the hearing aid user and non-hearing aid user at baseline. If the non-hearing aid user never acquired a hearing aid during the study, then after a period of ten years they were predicted to make 1.27 more MMSE errors than the woman who consistently used a hearing aid. There was also a significant level-2 correlation between the random effects for aid use and the intercept (ρ_{02}= -0.38, p = *.038,* 95% CI: -0.61, -0.08), indicating that participants reporting hearing aid use had a lower number of MMSE errors.

Of the covariates adjusted for in LMM 6, a greater number of MMSE errors were associated with old age, men, lower education, stroke, visual impairment and higher hearing thresholds. Although not a focus of this study, it is interesting to note that hypertension was actually predictive of fewer MMSE errors. There was also an interaction between age and time, indicating older adults had greater rates of change in MMSE errors compared to younger adults.

9.3.4 Residual Diagnostics

There are concerns that the MMSE data do not satisfy the assumption of residual normality in linear mixed models. The distributions of the residuals for the 5 random effects estimated by LMM 6 are shown in Figure 9.2. Both the level-1 residual and the random intercept have distributions that suggest positive (right-tail) skew. Further examination of level-1 residual diagnostic plots (Figure 9.3) further supports this interpretation. There are a large number of cases with residuals greater than 2 standard deviations above the mean but very few cases with residuals less than 2 standard deviations below the mean (Panel B). Further, normal probability plots deviate from the diagonal axis (Panel C, Panel D). It is therefore possible that the estimates are biased and may be unreliable so should be interpreted with caution.

9.3.5 GLZM

In an attempt to evaluate if the violations of normality resulted in biased estimates, generalized latent mixed models were also fitted to the same data. The formulas for these models are presented in the bottom rows of Table 9.2. Due to the extended time it took to run these models they were initially specified without random effects for Aid and Post-Aid slope. Smaller BIC and deviance values for the generalized latent mixed models suggested they were a better fit to the data compared to the linear mixed models (Table 9.3). Goodness of fit indices also indicated that the inclusion of a fixed effect for post-aid slope improved model fit. Thus, these analyses indicated that without adjusting for socio-demographics and health, hearing aid use was associated with fewer MMSE errors (IRR = 0.88, p = .001, 95% CI: 0.81, 0.95) and slower rates of change in MMSE errors (IRR = 0.97, p = .005, 95% CI: 0.95, 0.99). However, when adjusting for socio-demographics and health, the Post-Aid slope (rate of change in MMSE errors after the initial point of hearing aid use) was no longer significant (IRR = 0.98, p = .061, 95% CI: 0.96, 1.00) though the trend remained in the same direction and was close to significance levels.

Finally, unadjusted generalized mixed models (GLZMM 3, 4) that did include level-1 random effects for Aid and Post-Aid slope were tested. GLZMM 3 was found to have the best model fit of all models, suggesting that fixed and random effects for Aid should be retained. The inclusion of fixed and random effects for Post-Aid slope in GLZMM 4 did not improve model fit. Despite this, I report the parameter estimates from GLZMM 4 (see Table 9.4). Estimates were similar to those previously reported with significant fixed effects for Aid (IRR = 0.82, $p < .001$, 95% CI: 0.74, 0.92). The fixed effect for Post-Aid slope was not significant (IRR = 0.98, p =.083, 95% CI: 0.95, 1.00), though again trended in the same direction. As this final model took over 6 days

to converge, more complex analyses that made further adjustments for socio-demographics and health were not conducted.

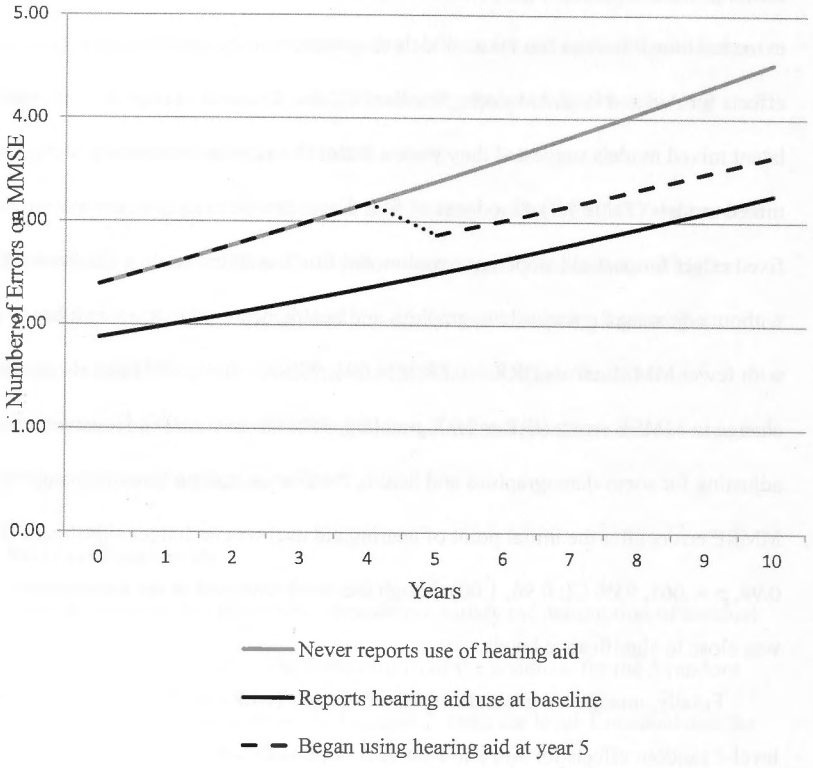
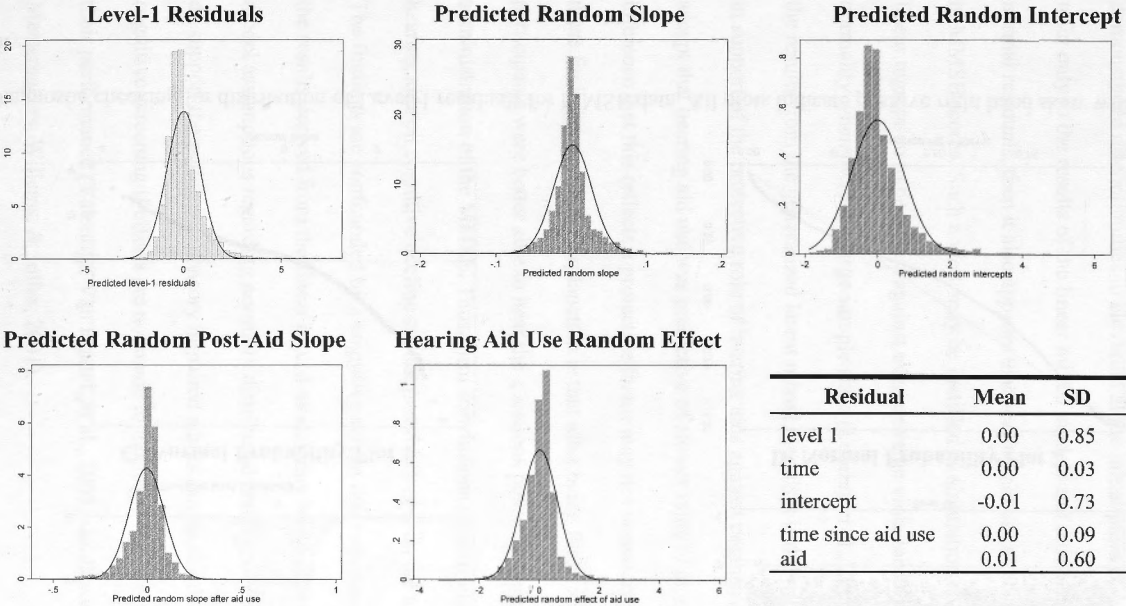


Figure 9.1 Predicted mean MMSE trajectories (errors) from LMM 6 for three hypothetical cases, each a woman aged 75 years at baseline with secondary schooling as their highest level of education, who across all waves reported no medical conditions (stroke, diabetes, heart attack, hypertension), had normal visual acuity, and a mild degree of hearing impairment at baseline (PTA=30 dB HL) with average rate of change in hearing (0.9dB per year). The only difference between the three hypothetical cases is their pattern of hearing aid use. Care should be taken when interpreting differences between slopes as these were not significant in generalized latent mixed models



Residual	Mean	SD	Skew
level 1	0.00	0.85	1.5
time	0.00	0.03	0.6
intercept	-0.01	0.73	1.7
time since aid use	0.00	0.09	-0.5
aid	0.01	0.60	1.0

Figure 9.2 Histograms of the level-2 random effect variance components and level-1 residual for Model 6 estimated by linear mixed model for MMSE change. Distributions indicate some deviation from normality.

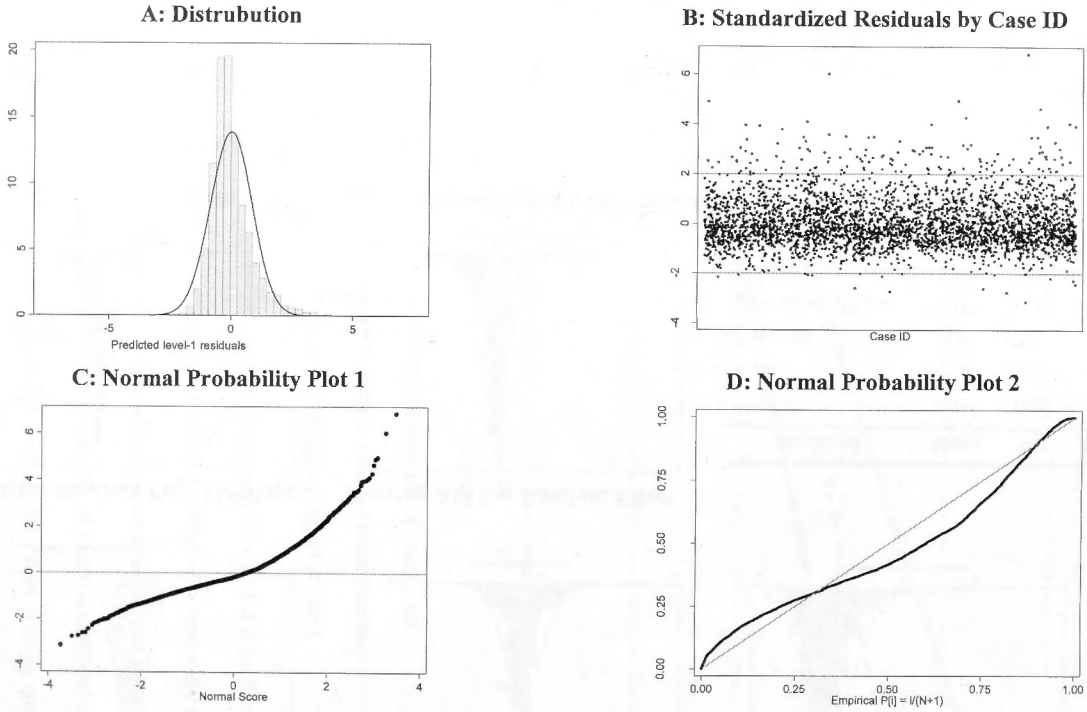


Figure 9.3 Diagnostic checking for distribution of Level-1 residuals for MMSE data. All plots indicate positive right hand skew with some deviation from normality.

9.4 Study 1 Discussion

This study aimed to investigate if performance on a general cognitive screening tool improved after participants began using hearing aids. The results clearly demonstrated discontinuities in elevation at the initial point of hearing aid use. If we refer only to the results of the linear mixed models and dismiss concerns about non-normal residuals, then it also appears that hearing aid use attenuated the rate of decline in MMSE scores. Such a view may be justified as simulation studies have demonstrated linear regression to be robust against even extreme violations of the assumption of normality when there are large sample sizes (Lumley et al., 2002). However, in light of the results from the generalized latent mixed models it is difficult to make a strong case in support of the protective role of hearing aids against cognitive decline. Even if we do accept that hearing aid use was predictive of slower rates of increase in MMSE errors, inference that this reflects a protective factor may not necessarily be the best account of these findings. A simpler explanation is that after being fitted with a hearing aid, participants were better able to hear the questions posed to them during the verbal administration of the MMSE. Thus, firm conclusions concerning the protective role of hearing aids on cognitive decline are not possible based on the analyses presented here. The findings are confounded by a cognitive screen that requires competent hearing, and the results derived from the linear mixed model may be unreliable due to violations of model assumptions regarding normally distributed residuals. Nevertheless, these results do support the notion that sensory impaired adults can be disadvantaged during cognitive screening if efforts are not made to minimise the effects of sensory losses on their performance (Valentijn, Van Boxtel, et al., 2005; van Boxtel, ten Tusscher, Metsemakers, Willems, & Jolles, 2001).

There are additional limitations to this study. Due to the wide intervals between waves, the exact point of hearing aid use is unknown. Further, the frequency of hearing

aid use was not investigated and it may be that more frequent hearing aid use is associated with higher levels of cognitive function and slower rates of decline. Finally, it is difficult to properly model a piecewise growth curve with only four waves of data. Some of these limitations are addressed in the following Study 2 which applies the same analytic approach to investigate discontinuities in trajectories of processing speed as a function of hearing aid use over 16 years of ALSA data.

9.5 Study 2 Methods

9.5.1 Study 2 Participants

This study uses the same ALSA sample as reported in Chapter 8 with the addition of a fifth wave of data. This fifth wave of data was collected in 2008 and was not included in previous chapters as it was unavailable when they were written, however it is included here to facilitate better estimation of a piecewise growth curve.

9.5.2 Study 2 Measures

The dependent variable was the Digit Symbol Substitution (DSS) test (Wechsler, 1981), which was used as a measure of processing speed. Time invariant independent variables were baseline age, sex and years of education. Time varying independent variables were PTA, MMSE score, CESD-D, number of medical conditions and hearing aid use. Hearing aid use was collected by the question "*Do you usually wear a hearing aid nowadays?*" with responses "*No*", "*Yes, Some of the time*" and "*Yes, all of the time*". These responses were dummy coded with '*No hearing aid use*' as the reference group. It was possible for participants to quit their hearing aid use, and also to change from being regular users to irregular users.

PTA was partitioned into two variables to facilitate the modelling of their within and between person effects on processing speed. By calculating the average score for each individual over all available waves, between-person effects of PTA were defined by person mean centering. Within-person effects were defined by the deviation between the person mean centred variable and their original time varying scores (Hoffman & Stawski, 2009). As in Study 1, two time variables were used, one for time in study (Time) and one for time using a hearing aid (Post-Aid Time).

9.5.3 Study 2 Analyses

Linear mixed models were used to analyse the 16 year change in DSS scores. As can be seen by the equations presented in Table 9.5, analyses followed a similar specification and sequence to Study 1. Firstly unconditional means and unconditional growth linear mixed models were fitted to 16 years of DSS data. Then a model of the within-person and between-person effects of hearing thresholds on processing speed was tested (Model 1). Frequency of aid use was then entered as a time varying fixed effect to evaluate differences in elevation (Model 2) and a fixed effect for time after initial hearing aid use was entered to evaluate discontinuities in trajectories (Model 3). A series of analyses that modelled hearing aid use as fixed and random effects were also conducted (e.g. Model 4). All analyses adjusted for the effects of baseline age, sex, years of education and number of medical conditions, as these covariates are not central to the substantive focus of this study they are not shown in the equations in Table 9.5 to conserve space.

Table 9.5 Level 1 and Level 2 equations

Model	Level 1*	Level 2**
Model 1	$DSS_{ij} = \pi_{0i} + \pi_{1i}Time_{ij} + \pi_{2i}PTA\_within_{ij} + \varepsilon_{ij}$	$\pi_{0i} = \gamma_{00} + \gamma_{01}Age_i + \gamma_{03}PTA\_between_i + \zeta_{0i}$ $\pi_{1i} = \gamma_{10} + \gamma_{11}Age_i + \zeta_{1i}$ $\pi_{2i} = \gamma_{20}$
Model 2	$DSS_{ij} = \pi_{0i} + \pi_{1i}Time_{ij} + \pi_{2i}PTA\_within_{ij} + \pi_{3i}Aid\_sometimes_{ij} + \pi_{4i}Aid\_always_{ij} + \varepsilon_{ij}$	$\pi_{0i} = \gamma_{00} + \gamma_{01}Age_i + \gamma\ PTA\_between_i + \zeta_{0i}$ $\pi_{1i} = \gamma_{10} + \gamma_{11}Age_i + \zeta_{1i}$ $\pi_{2i} = \gamma_{20}$ $\pi_{3i} = \gamma_{30}$ $\pi_{4i} = \gamma_{40}$
Model 3	$DSS_{ij} = \pi_{0i} + \pi_{1i}Time_{ij} + \pi_{2i}PTA_{within_{ij}} + \pi_{3i}Aid\_sometimes_{ij} + \pi_{4i}Aid\_always_{ij} + \pi_{5i}Time\_PostAid\_sometimes_{ij} + \pi_{6i}Time\_PostAid\_always_{ij} + \varepsilon_{ij}$	$\pi_{0i} = \gamma_{00} + \gamma_{01}Age_i + \gamma_{02}PTA\_between_i + \zeta_{0i}$ $\pi_{1i} = \gamma_{10} + \gamma_{11}Age_i + \zeta_{1i}$ $\pi_{2i} = \gamma_{20}$ $\pi_{3i} = \gamma_{30}$ $\pi_{4i} = \gamma_{40}$ $\pi_{5i} = \gamma_{50}$ $\pi_{6i} = \gamma_{60}$
Model 4	$DSS_{ij} = \pi_{0i} + \pi_{1i}Time_{ij} + \pi_{2i}PTA\_within_{ij} + \pi_{3i}Aid\_sometimes_{ij} + \pi_{4i}Aid\_always_{ij} + \pi_{5i}Time\_PostAid\_sometimes_{ij} + \pi_{6i}Time\_PostAid\_always_{ij} + \varepsilon_{ij}$	$\pi_{0i} = \gamma_{00} + \gamma_{01}Age_i + \gamma_{02}PTA\_between_i + \zeta_{0i}$ $\pi_{1i} = \gamma_{10} + \gamma_{11}Age_i + \zeta_{1i}$ $\pi_{2i} = \gamma_{20}$ $\pi_{3i} = \gamma_{30}$ $\pi_{4i} = \gamma_{40} + \zeta_{4i}$ $\pi_{5i} = \gamma_{50}$ $\pi_{6i} = \gamma_{60}$

*All Level 1 models include number of medical conditions.
**All Level 2 models also included fixed effects for sex, years of education and number of medical conditions.

Table 9.6 Frequency of hearing aid use at each wave of ALSA by sex and level of hearing loss, with mean age and DSS score.

	Total (n)	*New Users (n)	Men (%)	Mild HL (%)	Mod. HL (%)	Age		DSS	
						mean	(SD)	mean	(SD)
Wave 1									
Never used a hearing aid	928	-	47.3	44.1	15.0	78.0	(6.5)	30	(11)
No longer uses a hearing aid	84	-	61.8	43.3	47.4	80.6	(7.3)	25	(11)
Regular hearing aid use	154	-	64.0	22.5	76.0	81.4	(6.4)	28	(10)
Wave 3									
No aid	960	-	46.3	44.7	20.5	79.1	(6.1)	31	(11)
Occasional hearing aid use	115	89	63.5	29.4	69.2	82.0	(6.3)	26	(9)
Regular hearing aid use	92	12	58.0	6.9	93.1	83.3	(6.8)	26	(11)
Wave 6									
No aid	338	-	39.0	51.3	18.5	82.7	(4.9)	30	(11)
Occasional hearing aid use	53	36	55.6	28.8	69.5	85.0	(5.1)	30	(10)
Regular hearing aid use	51	32	44.8	10.3	89.7	85.8	(6.1)	27	(9)
Wave 7									
No aid	238	-	33.5	43.7	27.0	84.6	(4.4)	30	(11)
Occasional hearing aid use	53	34	50.0	33.9	64.3	85.8	(5.1)	28	(10)
Regular hearing aid use	40	16	44.0	20.9	79.1	86.2	(4.8)	26	(8)
Wave 9									
No aid	89	-	28.1	52.1	28.7	87.2	(3.5)	30	(10)
Occasional hearing aid use	28	16	50.0	27.6	72.4	87.9	(3.4)	28	(11)
Regular hearing aid use	26	16	40.0	3.9	96.2	89.7	(4.6)	28	(9)

Note: Mild Hearing Loss (HL) defined by 25dB < PTA < 40dB; Moderate HL defined by PTA > 40 dB; DSS: Digit Symbol Substitution test

*New Users are participants who did not report use of a hearing aid at the prior wave.

9.6 Study 2 Results

9.6.1 Hearing aid use by wave

There were 1547 participants observed an average 2.1 times in up to five waves over 16 years. Table 9.6 shows the number of hearing aid users at each wave with age, DSS and hearing loss descriptives. There was a greater proportion of hearing aid users at later waves. Notably, at all waves there were large numbers of participants with moderate levels of hearing loss who did not report use of hearing aid.

9.6.2 Within and between effects of PTA on DSS

Interclass correlation coefficients (ICC) indicated that the proportion of the total variability attributed to within-person variation was 61% for processing speed and 74% for hearing thresholds. After residualising the effects of age, sex, medical conditions and years of education, there were both within-person effects (γ_{02}=-0.12; $p < .001$; 95% CI: -0.17, -0.06) and between-person effects (γ_{03}= -0.07; $p < .001$; 95% CI: -0.11, -0.04) of PTA on DSS test scores. Higher person mean-centred hearing thresholds predicted lower levels of processing speed. Faster increases in hearing thresholds predicted faster rates of decline in processing speed. Post-hoc analyses also identified a slight interaction between-person mean centred PTA and within-person change in PTA ($\gamma_{04\ \text{between*within}} = 0.004$; $p = .046$; 95% CI: 0.001, 0.008) which suggested the association between within person change in hearing and decline in processing speed was more pronounced amongst those with poor overall hearing levels5. Older adults had lower DSS scores (γ_{01}= -0.740; p < .001; 95% CI: -0.821, -0.664) and faster rates of

[5] A second series of post-hoc analyses were stratified by hearing aid use. Interestingly, these found that the effects of within-person change in hearing thresholds on DSS scores were significant for non-hearing aid users (B=-0.15, $p < .001$, 95% CI: -0.21, -0.08, n=1332) but not for hearing aid users (B=-0.06, p =.224, 95% CI: -0.17, 0.04, n=376).

decline (γ_{11}= -0.033; p < .001; 95% CI: -0.052, -0.021). There were no sex differences in intercept of rates of decline.

9.6.3 Effects of Hearing Aid Use

Table 9.7 shows the goodness of fit indices for the competing models. Model 2 had the smallest BIC, AIC and -2ll deviance values, and so was considered to provide the best and most parsimonious fit to the data. This indicated that the fixed effect time-varying indicators of hearing aid use were reliable predictors of levels of processing speed. The inclusion of a second slope trajectory reflecting change in processing speed after the commencement of hearing aid use did not improve model fit, nor did the inclusion of a random effect for hearing aid use. Models that included random effects for irregular hearing aid use or random effects for post-aid slope did not converge.

Table 9.7 Goodness of fit for four mixed models for change in DSS scores (n = 1535).

Model	AIC	BIC	-211	(df)	Δχ2	(Δdf)	*p*	Comparison Model
Model 1	22672	22764	22643	(15)	-	-	-	-
Model 2	22665	22762	22633	(16)	10	(1)	.002	Model 1
Model 3	22670	22785	22632	(19)	1	(3)	.801	Model 2
Model 4	22675	22809	22631	(22)	1	(3)	.801	Model 3

Although Model 2 actually provided the best fit, the parameter estimates from Models 1, 3 and 4 are presented in Table 9.8 as they best address the aims of the study. I refer to the results of Model 4 in text. There was no significant association between occasional hearing aid use and DSS (γ_{20}=-0.02, p = .97; 95% CI: -1.07, 1.02), however regular hearing aid use did predict higher levels of DSS scores (γ_{30}=1.70, p = .02; 95% CI: 0.33,3.05). Although rates of change in DSS scores did tend to shift to more gradual

decline trajectories after the commencement of regular hearing aid use, this association was not significant and cannot be interpreted as indicating discontinuities in slope. The Model 3 estimated mean trajectories for four hypothetical individuals are illustrated in Figure 9.4. Of the covariates, age and education also predicted levels of DSS, however only age was associated with rates of change.

9.6.4 Study 2 Discussion

The aim of this study was to investigate discontinuities in levels and change in processing speed in relation to the regularity of hearing aid use. The key finding was that adults who reported regular hearing aid use had higher levels of processing speed whereas no benefit was observed for those who reported only occasional hearing aid use. Despite the discontinuity in levels, regular hearing aid use did not reliably alter subsequent rates of decline in processing speed. Thus, it does not appear that hearing aid use is protective against age-related decline in processing speed.

The findings regarding the association between hearing thresholds and processing speed are in line the results reported in Chapter 8, although they do not share the same interpretation. Chapter 8 demonstrated that hearing thresholds were leading predictors of subsequent rates of change in processing speed. In this study there were both within and between person effects of hearing thresholds on processing speed. Overall, people with poorer hearing were more likely to have poorer levels of processing speed, and as a participant's hearing acuity declined with time so too did their processing speed. Importantly, these effects remained after adjusting for age, health and socio-demographic variables. This may appear to conflict with Anstey, Hofer, & Luszcz (2003a) who reported no correlation between rates of change in processing speed and hearing over the first three waves of ALSA. These differences may be explained by the different methods used and extra two waves analysed in this study. Anstey et al (2003) were primarily interested in co-variation in intercepts and slopes estimated by

multivariate latent growth curves that included other cognitive domains and sensory. Their measure of hearing also comprised a latent factor that included all frequency thresholds (0.5-8kHz) for both left and right ear. In contrast, this study analysed a pure-tone average of 0.5, 1, 2, and 4 kHz in the better ear and used a different analytic approach.

9.6.4.1 Limitations

The final three waves are crucial for modelling a 2-piece growth curve, yet there were only 51 regular hearing aid users at wave 3 and even less at waves 4 and 5. Thus, analyses may have lacked the statistical power required to establish meaningful differences in rates of cognitive decline after the commencement of regular hearing aid. use.

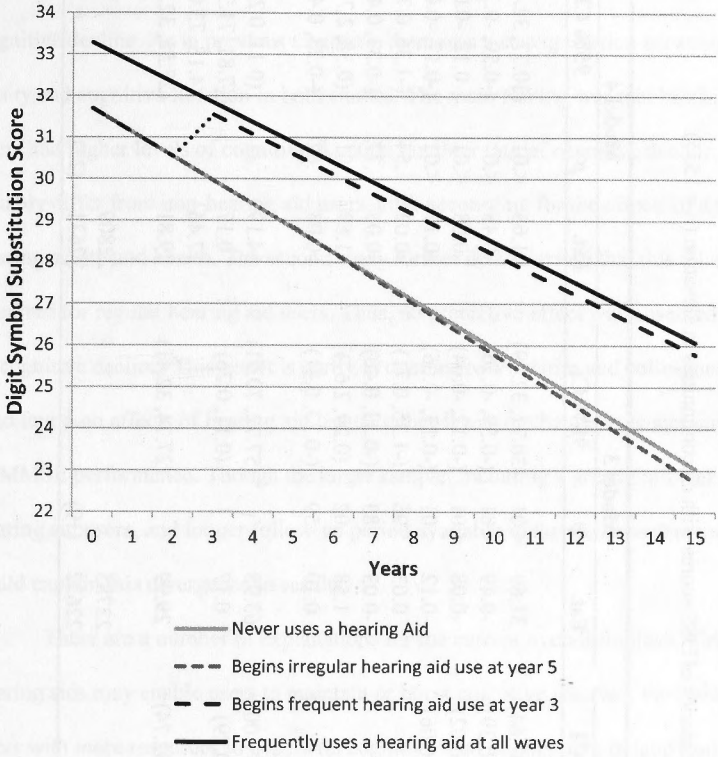


Figure 9.4 Model-X mean trajectories estimated from Model 4 for four hypothetical individuals who differ only in their pattern of hearing aid use. Each trajectory represents women aged 75 years at baseline with average rates of decline in hearing, who report no medical conditions and have 14 years of education. Frequent use of a hearing aid was associated with higher levels of processing speed compared to non-aid users and irregular aid users. Although frequent aid use may appear to predict mildly slower rates of decline in processing speed, this difference in slopes was not statistically significant.

Table 9.8 Estimates from linear mixed models of 16 year trajectories of DSS scores with discontinuities in elevation (n = 1535).

	Model 1			Model 3			Model 4		
	Est.	***p***	**95% CI**	**Est.**	***p***	**95% CI**	**Est.**	***p***	**95% CI**
Fixed Effects									
Intercept	31.80	<.01	(30.96, 32.64)	31.69	<.01	(30.77, 32.49)	31.63	<.01	(30.77, 32.49)
Slope (years)	-0.51	<.01	(-0.62, -0.39)	-0.49	<.01	(-0.59, -0.36)	-0.48	<.01	(-0.59, -0.36)
PTA between	-0.06	<.01	(-0.10, -0.02)	-0.08	<.01	(-0.12, -0.04)	-0.08	<.01	(-0.12, -0.04)
PTA within	-0.12	<.01	(-0.17, -0.06)	-0.12	<.01	(-0.17, -0.06)	-0.11	<.01	(-0.17, -0.06)
Aid(occasional)				0.03	.96	(-1.35, 0.71)	-0.03	.54	(-1.36, 0.71)
Post-Aid Slope(occasional)				-0.03	.83	(-0.33, 0.40)	-0.05	.77	(-0.33, 0.40)
Aid(regular)				1.58	.02	(0.29, 2.67)	1.69	.01	(0.38, 2.76)
Post-Aid Slope(regular)				0.10	.59	(-0.23, 0.41)	0.08	.62	(-0.23, 0.41)
Random Effects									
Intercept	64.04		(57.76, 71.00)	63.98		(57.72, 70.91)	64.19		(0.11, 0.26)
Slope	0.20		(0.13, 0.29)	0.17		(0.11, 0.26)	0.17		(7.81, 71.30)
Aid(regular use)							17.46		(4.18, 73.03)
Residual	30.28		(28.01, 32.74)	29.99		(27.74, 32.41)	29.81		(27.55, 32.26)
Model Fit									
BIC	22763			22785			22809		
-2ll (df)	-22642	(15)		-22632	(19)		-22631	(22)	

9.7 Conclusion

This chapter presented two studies investigating links between hearing aid use and cognitive decline. As in previous Chapters, there was a strong relation between hearing acuity and cognitive function in both studies. The main finding was that hearing aid users had higher levels of cognitive function but their rate of cognitive decline did not reliably differ from non-hearing aid users, after accounting for the effects of age, hearing acuity and health. The second study further demonstrated that this relation only occurred for regular hearing aid users. Thus, no protective effect was observed on rates of cognitive decline. This result is partly in contrast to Valentijn and colleagues (2005) who found no effects of hearing aid use on either levels or change in processing speed or MMSE performance. Though the larger sample, including a greater number of hearing aid users, and longer follow-up period available in the analyses presented here could explain this divergence in results.

There are a number of explanations for the current overall findings. Firstly, hearing aids may enable users to maintain or boost cognitive reserves, thus equipping users with more resources to cope with cognitive load or cognitive fatigue resulting from listening difficulties. According to the view that hearing aid use is akin to cognitive training, this could occur as a direct result of regular use supporting neuro-plasticity (Greenwood, 2007). Alternatively, reserves could be maintained indirectly through increased social engagement. The link between hearing aid use and cognition could in part be due to external factors such as education and occupational complexity, which are also believed to build cognitive reserve (Stern, 2009). Adults with higher socio-economic status and levels of education and may not only be more cognizant of their own hearing limitations, but also have greater awareness of hearing services available to them and find these services to be more affordable.

The correlational design and reliance on observational data means that reciprocal causation is a major constraint on the interpretation of these findings. It is not possible to infer a directional causal relation between hearing aid use and cognitive function. Individual differences in cognitive function have been proposed as a factor that may explain why adults with similar patterns of hearing loss have different listening experiences after being fitted with identical hearing aids (Pichora-Fuller, 2009). Cognitive function has been shown to be an important factor in a person's ability to properly use a hearing aid (Lunner, 2003) and people with higher cognitive functioning derive greater benefit from their use (Gatehouse, Naylor, & Elberling, 2003). Cognitive impairment has been shown to be a predictor of low hearing aid utilisation among adults identified in need of a hearing aid (Lopez-Torres Hidalgo et al., 2009). Further, it can take around 3 months to become accustomed to hearing aids after an initial fitting (Weinstein, 2000) and functions such as processing speed are known to play an important role in acclimatizing and learning how to comfortably use hearing aids. People with poor cognitive function may therefore find it harder to adapt to a new hearing environment, while those with greater cognitive resources may be better equipped to make the most of any reductions in cognitive load (Beck, 2011). Thus, we should not dismiss the possibility that a causal pathway between hearing aid use and processing speed is in the opposite direction to that suggested by the analyses presented in this chapter.

From a clinical perspective, hearing aid use should not be considered a panacea to alleviating adverse effects of hearing loss (Lin, 2012). It is important to acknowledge that auditory rehabilitation will have greater benefits when integrated with counselling, training and proper follow-up of hearing aid fitting. Lin (2012) also describes other hearing assisting technologies, such as cochlea implants and hearing loop induction

systems that should be considered when designing a treatment plan for adults with hearing loss.

There are limitations that are common to both studies presented in this chapter. The long time frame between waves, particularly between wave 3 and wave 6 of ALSA, makes it difficult to precisely model trajectories over these intervals and the exact time at which participants began using hearings aids could not be determined. Hearing aid use was defined by participant self-report and it was not possible check that aids were fitted correctly, or control the type or specification of hearing aids. A recent review of hearing aid research found that synthesis was hampered by inconsistencies in measures of hearing aid usage. Thus, standardized approaches to collecting hearing aid information for research purposes are needed (Perez & Edmonds, 2012). The two cognitive measures used throughout this thesis reflect a screen for general impairment and a processing speed. However, this ignores a number of domain general and hearing specific cognitive processes that are heavily involved in auditory functions. Other general cognitive processes implicated in listening tasks, include working memory, inhibition and attention (Pichora-Fuller & Singh, 2006; Rönnberg, Rudner, & Zekveld, 2009).

In summary, frequent and persistent hearing aid use predicts better cognitive functioning but does not slow the progression of cognitive decline. Future research should include other cognitive domains and ideally obtain more thorough measures of hearing aid type and usage. While this chapter applied a naturalistic quasi-experimental design with observational data, ultimately the best way to evaluate if hearing aids can hinder cognitive decline will be to apply long-term clinical trial designs with large samples (Lin, 2012).

CHAPTER 10: Summary

Synopsis

This chapter provides a brief summary of the research presented in the previous chapters, highlighting key points, acknowledges limitations and outlines avenues for future research on hearing-related cognitive decline.

10.1 Introduction

This thesis was primarily concerned with documenting the level of Hearing Loss among older Australians and exploring longitudinal inter-relationships between hearing and cognitive function. Thus, the presented research was guided by two themes, an epidemiological perspective which focused on sensory and cognitive impairment, and a psychological perspective which drew upon cognitive ageing literature and focused on degree of functioning in healthy adults. An epidemiological perspective is important because it describes the extent of these losses at a population level, allowing researchers, clinicians and policy analysts to understand the burden of, and evaluate risk factors for sensory and cognitive impairment. This differs from a psychological perspective, which is more focused at the level of the individual and seeks to understand inter-relations between domains.

10.1.1 Population Estimates

Chapters 4-5 primarily addressed the epidemiological aims of this thesis. In these chapters, hearing loss was reported to be highly prevalent among older age groups, and its co-occurrence with vision loss and cognitive impairment was also considerable. The unique contribution of this section was to provide estimates for narrow age groups, estimate transition rates into sensory loss and calculate the number of years adults could expect to live with sensory impairment in the years before death. One of the more interesting findings of this largely descriptive work was to draw attention to sex differences. Hearing loss is often perceived to be a men's health issue, but this was not shown to be the case for older age groups. For adults older than 80 years of age, hearing loss affected men and women in equal numbers and women actually lived for longer periods with hearing loss. Further, hearing thresholds were also unique predictors of increased mortality risk for women but not for men.

Self-report data were not found to be reliable proxies for audiometric hearing loss in epidemiological research as they were insensitive to age trends and influenced by social norms. Their main value is presumed to be in assessing hearing disability, though this was not explicitly tested here.

10.1.2 Predicting Change in Hearing Thresholds

Chapter 7 investigated predictors of level and rates of change in hearing thresholds. The purpose of this was to identify risk factors for actual hearing decline, rather than hearing impairment. Firstly, this study replicated existing descriptions of age-related hearing loss, revealing greater rates of change for higher frequencies across all ages, and faster decline in old age. Notably sex differences for lower range frequencies were not observed, though men generally had lower overall hearing levels. The main finding was that there were few predictors of rates of change in hearing thresholds, these included age, cognitive impairment, noise exposure and hypertension. Interestingly, these latter two factors predicted more gradual rates of change, most likely because the level of hearing function for these groups was already low. Cognitive impairment predicted faster rates of change in hearing thresholds, and on occasions when a participant was identified with possible cognitive impairment there was also a corresponding rise in levels of hearing thresholds.

10.1.3 Hearing-related Cognitive Decline

If 'age' is an empty variable that serves as a proxy for other developmental processes, then the concept of 'age-related cognitive decline' may be a misnomer. It is therefore incumbent upon developmental researchers to identify those processes that that provide greater insight into decline of fluid abilities. To this end, the latter chapters of this thesis were chiefly concerned with hearing-related cognitive decline. Chapter 8

explored time-ordered interrelations between hearing thresholds and processing speed. This revealed a longitudinal link between cognition and hearing, but in the opposite direction to the findings of Chapter 7. There was clearly a dynamic association whereby hearing was a leading indicator of subsequent cognitive decline. This is consistent with cascade models and biomarker accounts of cognitive ageing. However, a common cause hypothesis remains a plausible explanation and none of these theories are mutually exclusive. It is possible that hearing-related cognitive decline may be the result of generalised deterioration of the central nervous system, or mediated by reduced engagement and increased cognitive load that arise from communication difficulties. The final results chapter of this thesis attempted to test the latter explanation by examining the role of hearing aids.

10.1.4 Hearing Aids and Cognitive Function

One of the main aims of this thesis was to examine the hypothesis that hearing aids may protect against cognitive decline. To this end, Chapter 9 reported on a naturalistic intervention that investigated levels and rates of change in cognitive function before and after the uptake of hearing aids. Despite the high prevalence of hearing loss, hearing aid utilisation was low, with only half of those with moderate levels of hearing loss reporting use of an aid. Hearing aid use predicted higher levels of cognitive function, but not slower rates of change. This is not entirely consistent with the proposition that aids are protective against cognitive decline; rather it is more likely that those with intact cognition were better equipped to make the transition to using a hearing aid. Indeed, screening of hearing impairment in adults aged 55-75, and early take-up of hearing aids by those with PTA > 30 dB HL has been shown to maximise the benefits of their use (Davis et al., 2007). It is likely that early adopters are more successful at becoming comfortable with hearing aids and have greater retention rates as they have greater capacity to acclimatize to a new auditory environment at a time when

they are under less cognitive load. Although not specifically tested in this thesis, it can be surmised that early take-up of hearing aids will improve quality of life (Davis et al., 2007) and should therefore be considered effective tools for compressing morbidity. However, some researchers have suggested that uptake of hearing aids at a time when there are no real benefits could be detrimental (Gopinath et al., 2011), so a balance must be struck between commencing hearing aid use too early or too late.

10.1.4.1 Hearing aid costs in Australia

One potential barrier to hearing uptake is cost and awareness of hearing services. Hearing assessment and rehabilitation programs are fully subsidised to pensioners and veterans in Australia under the Australian Government Hearing Services Program (Australian Government Department of Health and Ageing, 2012a). Currently, hearing aids are made available free of charge to eligible Australians if their 3 frequency (0.5, 1, 2 kHz) average hearing loss exceeds 23dB. However, users must pay annual maintenance fees and top-up costs should they choose a more advanced device over the base model provided. Eligible adults who do not have this minimal level of hearing loss may still access subsidised hearing programs if they are motivated to wear a hearing aid device and meet one of the following criteria (Australian Government Department of Health and Ageing, 2012b):

- Untreatable visual impairment that hinders lip reading
- High frequency (2, 3, and 4 kHz) hearing loss greater than 40 dB
- Presence of tinnitus
- Previous use of a hearing aid

For Australians who are not eligible for the Australian Government Hearing Services Program, private health insurers often cover up to $1000 for hearing aids and a tax rebate of 20% is available for expenses in excess of $1500. However, this does not

cover the full costs of a hearing aid in Australia, which depending on the model and specifications range between $2000 and $4000 per aid. Additional costs may also be needed to meet other hearing rehabilitation services, and hearing aid batteries are also expensive making them a considerable financial burden (Easton, 2012; Gopinath et al., 2011).

Prevalence presented in Chapter 4 showed that 59% of adults aged over 65 had at least a mild degree of hearing loss. To remain consistent with standard definitions of hearing loss, this thesis did not estimate prevalence of 3 frequency averages currently used as a criterion for hearing aid subsidy. In June 2011 the resident population of Australia aged 65 years and older was estimated to be 3,076,539 persons (Australian Bureau of Statistics, 2012a) of which 1.8 million were likely to have at least a mild degree of hearing loss based on the prevalence provided in Chapter 4. The higher threshold cut-off of 25 dB and inclusion of 4kHz frequencies means that these prevalence estimates can be considered conservative estimate of 3 frequency definitions of hearing loss.

10.1.5 Lessons Learned from Methodology of Harmonised Data

The key advantage of harmonisation and data pooling for the purposes of this thesis was in providing population estimates that could be generalized beyond the individually sampled populations and increased statistical power. This was most evident in Chapters 4-6. An additional benefit was found in the cross-institutional and interdisciplinary collaboration that enabled me to leverage the expertise of colleagues with backgrounds in clinical ophthalmology, epidemiology, statistics, demography, psychology gerontology.

Unfortunately, these advantages were minimized because few DYNOPTA studies collected measures that could be brought to bear on the substantive questions posed by this thesis. However, the benefits of combining studies were evident in other

DYNOPTA projects which I was involved in, including projects reporting prevalence estimates of probable dementia (Anstey et. al., 2010), medical conditions (Bielak, Byles, Luszcz, & Anstey, 2012) and norms for neuropsychological tests (Kiely et. al., 2011). By combining studies from different licensing jurisdictions, the effect of differing age-based assessments on driving rates could be made (Ross et al., 2009; Ross, Browning, Luszcz, Mitchell, & Anstey, 2011). And the value was DYNOPTA was also demonstrated by documenting the lack of longitudinal data on Indigenous Australians in population based studies (Anstey, Kiely, et al., 2011). This is an important issue as Aboriginal and Torres Strait Islanders have considerably lower life expectancies and poorer health outcomes compared to non-indigenous Australians.

Obviously, unless studies are designed to be pooled from the outset, or share equivalent variables and common sample characteristics, then testing of more refined and complicated pathways is better suited to single study analyses. Although the DYNOPTA studies shared many characteristics, variable harmonization often diluted variables. This reduced variability and often resulted in data that was appropriate for cause grained analyses only. Despite this, the value of combining studies should not be dismissed as such broad indices documenting the health of a population are informative from a public health and policy perspective.

10.1.6 Limitations

Many conceptual and methodological limitations were noted throughout this thesis as they arose, however a few general caveats warrant repeating here.

10.1.6.1 Time schedule

As noted in each chapter there were large time spans between observations, sometime up to 6 years. Thus short term changes in cognition and hearings functioning could not be assessed and transition rates reported in early chapters are likely to be

imprecise. The baseline data also dates back to the early 1990's and it is unclear how well the sample profile maps onto the current and future cohorts of older Australians.

The longitudinal models employed in this thesis used either age or time in study as the time metric. There is a view that these time metrics are poor temporal indices of ageing processes and other metrics such as time to event (Sliwinski & Mogle, 2008; Sliwinski, Hofer, Hall, Buschke, & Lipton, 2003) or distance to death (Diehr, Williamson, Burke, & Psaty, 2002; Gerstorf, Ram, Ghisletta, et al., 2008) are more informative as they provide an insight into morbidity and mortality related processes.

10.1.6.2 Missing data

Attrition and non-response are common sources of bias in longitudinal studies of ageing. They are associated with old age, male gender, poor health, cognitive impairment and low education (Anstey, Hofer, & Luszcz, 2003b; Chatfield et al., 2004; Matthews et al., 2004). In this thesis, generally missing data was assumed to be missing at random (MAR) (Little & Rubin, 2002) and addressed in structural equation models using full information maximum likelihood. Other options that were not adopted in this thesis were use of multiple-imputation for non-response and adjusting for attrition using pattern mixture techniques or longitudinal weights. It is acknowledged that the findings presented here are possibly biased towards healthy older Australians.

10.1.6.3 Observational survey design

This thesis relied exclusively on observational survey and performance data, and the correlational design meant I could only explore associations between the factors of interest. It was therefore not possible to draw definitive conclusions regarding causal links between hearing and cognitive decline. Ultimately experimental designs and randomised trials are best placed to examine causative pathways and protective effects of hearing aids on cognitive function (Lin, 2012).

10.1.6.4 Definition of cognitive impairment

Cognitive impairment was defined by a score of 23 or lower on the MMSE (Folstein et al., 1975a). Whilst the MMSE is a commonly applied measure in cognitive epidemiology research, I do recognise it is a screen of cognitive dysfunction and not a clinical diagnosis of a particular neurocognitive disorder. Care must therefore be taken not to draw too strong a conclusion regarding specific neurological conditions like Alzheimer's Disease. Nevertheless, as a screen of possible cognitive impairment the MMSE has does have reasonable sensitivity and specificity for detecting dementia and related disorders and its common use means results can easily be interpreted by a wide audience and are more easily comparable with other studies.

10.1.6.5 Other aspects of hearing and cognition

This thesis has only considered hearing measures that were self-reported, categorical indicators of hearing loss, and averaged thresholds in the better ear. Also, only two measures of cognitive function were included. This was due to data limitations and the measures available, space constraints and the key role processing speed is considered to play in cognitive ageing. All of these measures operationalise hearing on a single dimension. However, age-related hearing loss is not a unitary construct. For example, hearing loss may be unilateral or bilateral, localised to high range or low range frequencies, and audiograms can be characterised by concave or convex gradients. Thus a number of different patterns of age related hearing loss can be identified and are recognised by audiologists (Weinstein, 2000). It is possible that these each have specific risk-factors and varying implications for older adults functioning. This is evident in audiogram notches of people with a history of noise exposure. There are some studies that do demonstrate a degree of specificity between predictors of peripheral hearing and particular features of audiogram shape (Demeester et al., 2010; Demeester et al., 2009; Hwang, Wu, Hsu, Liu, & Yang, 2009; Jerger, Chmiel, Stach, & Spretnjak, 1993).

Scientific knowledge of hearing loss will be broadened by research that distinguishes between these different audiogram characteristics. Similarly, it would be insightful to explore other aspects of non-peripheral hearing abilities and cognitive factors not covered by this thesis. For example, there is a view that links between sensory functioning and cognition are strongest for vision and episodic memory (Anstey, 2011; Anstey et al., 2003a). It would also be informative to also investigate links between cognitive function and central auditory processes.

10.1.7 Implications and Concluding Thoughts

Our abilities to see, hear and think are critical abilities that underlie our capacity to perceive, understand, and interact with the environment in which we live. Whilst functional modalities of cognition and sensation are often treated as distinct areas of enquiry, they are intricately linked and scientists are increasingly focusing on how they interact. The importance of the integration between hearing and cognition is particularly evident in the study of late life human development. Concomitant loss of these abilities in older adulthood is devastating to functional independence, especially for those who have enjoyed high functioning throughout their lifespan. Peripheral hearing ability could be a useful marker of cognitive decline and biological age. The demographic transition towards an ageing population will also translate to a greater impact of age-related hearing loss on society. It may be possible to protect against hearing loss via modifiable risk-factors to a degree, but it will nevertheless decline with age. Ultimately a better appreciation of the impacts of hearing loss and greater adoption of hearing assistive technologies will also go a long way towards mitigating its burden. A more nuanced understanding of long-term interactions between hearing, listening, cognition and hearing aids is therefore needed.

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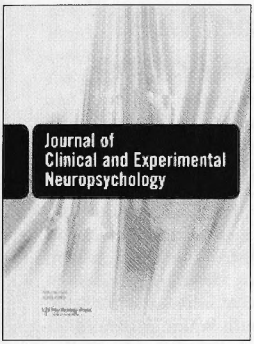
Appendix I: Published Articles



Journal of Clinical and Experimental Neuropsychology
Publication details, including instructions for authors and subscription information:
http://www.informaworld.com/smpp/title~content=t713657736

Functional equivalence of the National Adult Reading Test (NART) and Schonell reading tests and NART norms in the Dynamic Analyses to Optimise Ageing (DYNOPTA) project

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First published on: 04 December 2010

To cite this Article Kiely, Kim M. , Luszcz, Mary A. , Piguet, Olivier , Christensen, Helen , Bennett, Hayley and Anstey, Kaarin J.(2010) 'Functional equivalence of the National Adult Reading Test (NART) and Schonell reading tests and NART norms in the Dynamic Analyses to Optimise Ageing (DYNOPTA) project', Journal of Clinical and Experimental Neuropsychology,, First published on: 04 December 2010 (iFirst)
To link to this Article: DOI: 10.1080/13803395.2010.527321
URL: http://dx.doi.org/10.1080/13803395.2010.527321

JOURNAL OF CLINICAL AND EXPERIMENTAL NEUROPSYCHOLOGY
2010, i*First*, 1–12

Psychology Press
Taylor & Francis Group

Functional equivalence of the National Adult Reading Test (NART) and Schonell reading tests and NART norms in the Dynamic Analyses to Optimise Ageing (DYNOPTA) project

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This study investigates the functional equivalence of two measures of irregular word pronunciation—National Adult Reading Test (NART) and Schonell—which are popular instruments used to assess verbal neurocognitive functioning and to estimate premorbid IQ. We report norms for the NART in a pooled sample from 3 Australian population-based studies of adults aged 65–103 years. Norms were stratified by sex and age left school in 5-year age groups. The NART and the Schonell had a strong linear relation, allowing for the imputation of NART scores based on Schonell performance within 1 study. Neither measure was sensitive to the effects of sex after adjusting for the effects of age and education. Early school leavers performed worse on both measures. Data pooling enables greater precision and improved generalizability of NART norms than do methods that use single older adult samples.

Keywords: Verbal abilities; Harmonization; Premorbid IQ; Crystallized intelligence; National Adult Reading Test; Norms; Dynamic Analyses to Optimise Ageing; Australian Longitudinal Study of Ageing; Canberra Longitudinal Study of Ageing; Sydney Older Persons Study.

Investments in large-scale studies of neuropsychological function have underwritten the current movement towards a greater synthesis and optimal use of existing data sources within ageing research disciplines. One strategy to achieve synthesis is data pooling and variable harmonization. Pooling independently designed studies enables analyses of large datasets, increases statistical power to investigate low-prevalence disorders, aids comparative cross-population research, and provides for simultaneous replication of empirical findings (Anstey et al., 2010; Hofer & Piccinin, 2009; van Buuren, Eyres, Tennant, & Hopman-Rock, 2005). Data

The data on which this research is based were drawn from several Australian longitudinal studies including: the Australian Longitudinal Study of Ageing (ALSA), the Canberra Longitudinal Study of Ageing (CLS), and the Sydney Older Persons Study (SOPS). These studies were pooled and harmonized for the Dynamic Analyses to Optimise Ageing (DYNOPTA) project. DYNOPTA was funded by National Health and Medical Research Council (NHMRC) Grant 410215. O.P. is supported by NHMRC Clinical Career Development Award Fellowship 510184, K.A. is supported by NHMRC Fellowship 366756, and H.C. is supported by NHMRC Fellowship 525411. All studies would like to thank the participants for volunteering their time to be involved in the respective studies. Details of all studies contributing data to DYNOPTA, including individual study leaders and funding sources, are available on the DYNOPTA website (http://dynopta.anu.edu.au).The findings and views reported in this paper are those of the authors and not those of the original studies or their respective funding agencies.

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http://www.psypress.com/jcen
DOI: 10.1080/13803395.2010.527321

pooling is particularly useful for the generation of norms, which otherwise are often based on small sample studies with restricted population coverage (e.g., Tombaugh & Hubiey, 1997; Uttl, 2002). Although the notion of multistudy analysis via data pooling or meta-analysis is not new, often pooled studies will not share operationally identical measures. In these instances, data pooling is contingent upon the existence of measures that index the same (though not identical) theoretical construct and so are functionally equivalent. A growing literature explicitly addresses the use of harmonization methods for combining functionally equivalent measures to facilitate multistudy analyses (McArdle, Grimm, Hamagami, Bowles, & Meredith, 2009; Pommerich & Dorans, 2004; van Buuren et al., 2005). Research on the harmonization of cognitive measures is a relatively recent endeavor despite the more developed practice of data pooling of biological variables, such as serum cholesterol, in other epidemiological and medical disciplines (e.g., Dyer, 1986).

Both the Schonell Graded Word Reading Test ("Schonell"; Schonell, 1942) and the National Adult Reading Test (NART; Nelson, 1982) are 50-item graded pronunciation tasks for irregularly pronounced words. They each assess verbal abilities and have been used as a proxy for premorbid verbal IQ (VIQ) and full-scale IQ (FSIQ) in adults (Crawford, Deary, Starr, & Whalley, 2001; Crawford, Parker, Stewart, Besson, & De Lacey, 1989; Nelson & McKenna, 1975) based on the Wechsler Adult Intelligent Scale (WAIS; Wechsler, 1955). The Schonell, however, was originally developed to assess reading ability in children and is therefore relatively insensitive to reading ability in well-educated adults (Franzen, 2000). In order to provide more accurate indexation of premorbid IQ, the NART was developed and standardized in an adult population in Britain (Nelson, 1982; Nelson & McKenna, 1975) and has since been revised (NART–2) for the Wechsler Adult Intelligence Scale–Revised (WAIS–R) and for North American populations (American National Adult Reading Test, AMNART; North American Adult Reading Test, NAART). Short forms have also been developed (Strauss, Sherman, & Spreen, 2006).

The NART has robust reliability, sound psychometric properties, and brief administration time, and it is resistant against cognitive decline except in severe cases or in the presence of focal reading disorders or semantic dementia. Reflecting their strong indexation of putative crystallized abilities (Horn & Cattell, 1967), longitudinal analyses of word pronunciation tasks have revealed their relative stability in late life, typically showing only slight age-related declines in performance with no individual differences in intraindividual variability (Anstey, Hofer, & Luszcz, 2003). For these reasons, the NART is commonly used as a proxy measure of premorbid IQ in large population-based studies used in epidemiological and cognitive neurobiological research (e.g., Anstey et al., 2003; Christensen et al., 1999; Luszcz, Bryan, & Kent, 1997).

The NART and the Schonell are functionally equivalent measures that share similar task parameters and differ predominantly in item difficulty. Research investigating their shared psychometric properties has primarily been in relation to predicting WAIS IQ. Crawford et al. (1989), for example, investigated whether combining the NART and Schonell tasks would provide increased prediction accuracy of IQ for subjects with limited verbal abilities (i.e., NART items correct <10). They reported, however, that doing so systematically underestimated WAIS IQ and resulted in a nonlinear relationship between predicted and observed WAIS IQ. There has been little research investigating the direct association and compatibility between these two measures in nonclinical populations.

The available norms for the NART itself are limited. NART norms have not been derived from random samples of the general population, but rather have been generated through recruitment via interest groups, advertisements, and convenience samples. Sample size restrictions have also necessitated the reporting of normative data for the NART in overlapping 10-year age groups, with typically small cell counts for older age groups (e.g., Spreen & Strauss, 1998; Uttl, 2002). For example, norms reported for adults between the ages of 50 and 100 years, stratified by 5-year age groups, sex, and a binary measure of education, would result in 40 subgroups. The likelihood of low cell counts in these subgroups brings into question the generalizability of reported norms, particularly for older age groups, and undermines the validity of regression-based norms that adjust for sociodemographic variables (Fastenau, 1998). Moreover, NART norms specific to the Australian population do not exist. Currently, Australian clinicians must use UK NART norms, which may be biased by cultural differences in curriculum, vernacular, and pronunciation (Mathias, Bowden, & Barrett-Woodbridge, 2007).

Objectives

The objectives of this study were twofold. The first aim was to assess the functional equivalence of NART and Schonell tasks and the capacity to produce comparable predicted NART scores based on Schonell performance within a single study where baseline NART scores were not obtained. The second aim was to perform multistudy comparisons across three large population-based studies that have obtained NART data. The baseline data from these studies were used to produce normative NART data based on a sample of over 2,636 older Australians, producing greater subgroup precision than previously reported. The relations between the NART and contextual variables were also explored.

METHOD

Study design

The data were drawn from the Dynamic Analyses to Optimise Ageing (DYNOPTA) project (Anstey et al.,

2010) dataset, which has harmonized and pooled data from nine Australian longitudinal studies of ageing. Three studies within DYNOPTA administered either the NART or the Schonell. Both the Australian Longitudinal Study of Ageing (ALSA; Luszcz et al., 2007) and the Canberra Longitudinal Study (CLS; Christensen et al., 2004) have repeated measures of the NART on up to four occasions. The Sydney Older Person Study (SOPS) included the Schonell at both baseline and first follow-up, whereas the NART was only included at the first follow-up assessment. Baseline NART scores are therefore study censored for SOPS participants. Study censoring refers to data that were not obtained by a particular study. This is a common problem encountered in data pooling. Because the NART was not administered at baseline, the mechanism for study-censored data is known and can be classified entirely as Missing at Random (MAR; Little & Rubin, 2002; Salthouse, 2004). The first study aim relates to predicting baseline NART scores for this study-censored population by imputation procedures that are appropriate for data that are assumed MAR.

Contributing study sample profiles

The study-censored SOPS sample consisted of a random sample of community-dwelling residents aged 75 years and older from eight local government areas from the inner west of Sydney. The two-stage sampling design included 327 World War II veterans and widows and 320 nonveterans. Because of the selection process, 17 participants were sampled in both sampling frames (Piguet et al., 2003). Clinical interview and cognitive assessments were conducted by a trained physician experienced in geriatric medicine. The investigation of the functional equivalence between the NART and Schonell is based on data from the first follow-up wave when each measure was obtained concurrently in SOPS.

Within SOPS the baseline measurement wave occurred between August of 1991 and September of 1993 and included 630 participants (females = 50.5%) with a mean age of 80.5 years (SD = 4.2). The average time interval between baseline and the first follow-up wave was 2.9 years (SD = 0.3). A total of 128 participants died before the start of the first follow-up wave; 448 respondents (female = 54.7%) participated in the first follow-up wave, resulting in an attrition rate of 12.9%. The average school leaving age was 14.6 years (SD = 1.56), with 55% of participants leaving school before the age of 15 years.

The Australian Longitudinal Study of Ageing (ALSA; Luszcz et al., 2007) randomly sampled both community dwellers and residents in aged care, aged over 70, from 20 local government areas from metropolitan Adelaide. Coresident partners aged over 65 years and other coresidents over 70 were also invited to participate. The Canberra Longitudinal Study (CLS; Christensen et al., 2004) randomly sampled community-dwelling residents aged 70 years from the Australian Capital Territory and Queanbeyan.

Pooled sample profile

Pooled baseline data from the three studies were used to generate NART norms, which are based on observed NART scores for ALSA and CLS and predicted NART scores for SOPS. The pooled sample (see Table 1) comprised 2,636 participants (female = 51.1%). Of these, 48.2% left secondary school at the age of 14 years or younger, 73% were born in Australia, and 95% reported English as their first language. The average age was 77.8 years (SD = 5.9, range: 64–103), and average score on the Mini-Mental State Examination (MMSE) was 27.1 (SD = 2.9). Baseline waves from the contributing studies were contemporaneous, and NART data were obtained between the years 1990 and 1993.

Measures

In SOPS, the Schonell was administered at both baseline and the first follow-up wave, with the NART being administered in the first follow-up wave only. The ALSA and the CLS each administered the NART on four measurement occasions; however, only baseline NART scores are used for the generation of norms. The NART and Schonell were each scored as the number of words correctly pronounced and ranged between 0 and 50.

Time-invariant covariates were obtained at baseline and included "sex" (0 = male, 1 = female), "language first spoken" (0 = English, 1 = other), and "age left school." A continuous measure of age left school was used to test whether education mediated the relation between the NART and Schonell in SOPS. To ensure commonality across studies when reporting norms, a harmonized variable for "age left school" was collapsed to a binary coding (0 = 14 years or younger; 1 = 15 years or older). Time-varying covariates included "age at time of observation" and MMSE scores (Folstein, Folstein, & McHugh, 1975), which were used as an index of cognitive status. Covariates were chosen as candidates for inclusion in the imputation model because of their documented relations with the NART and Schonell.

Analysis

Imputation of NART scores in SOPS

Baseline NART scores were imputed by conditional ordinary least squares mean imputation (Little & Rubin 2002; McKnight, McKnight, Sidani, & Figueredo, 2007). Bivariate Pearson correlations were used to determine association between the Schonell, NART, and candidate covariates. Covariates were mean centered to minimize multicollinearity. Backwards hierarchical regression was used to identify predictor covariates for inclusion in the imputation model, with both linear and nonlinear effects. Backwards regression is appropriate because the aim of this exploratory analysis is to identify the optimal model for predicting NART scores, rather than hypothesis testing. The NART was not imputed for cases

TABLE 1
Pooled sample profile

	Age group (years)						
	<70 (n = 96) n (%)	*70–74 (n = 740) n (%)*	*75–79 (n = 839) n (%)*	*80–84 (n = 577) n (%)*	*85–89 (n = 286) n (%)*	*90–94 (n = 80) n (%)*	*≥ 95 (n = 18) n (%)*
Sex							
Males	10 (0.00)	387 (0.15)	452 (0.17)	299 (0.11)	161 (0.06)	34 (0.01)	4 (0.00)
Females	86 (0.03)	353 (0.13)	387 (0.15)	278 (0.11)	125 (0.05)	46 (0.02)	14 (0.01)
City							
Adelaide	96 (0.04)	369 (0.14)	335 (0.13)	252 (0.10)	170 (0.06)	35 (0.01)	7 (0.00)
Canberra	0 (0.00)	371 (0.14)	267 (0.10)	171 (0.06)	59 (0.02)	32 (0.01)	7 (0.00)
Sydney	0 (0.00)	0 (0.00)	237 (0.09)	154 (0.06)	57 (0.02)	13 (0.00)	4 (0.00)
Career occupation							
Managers and professionals	7 (0.00)	203 (0.01)	204 (0.10)	140 (0.07)	77 (0.04)	20 (0.01)	2 (0.00)
Clerical and associate professional	18 (0.01)	155 (0.07)	134 (0.06)	86 (0.04)	37 (0.02)	14 (0.01)	4 (0.00)
Tradespersons	14 (0.01)	107 (0.05)	103 (0.05)	92 (0.04)	46 (0.02)	5 (0.00)	1 (0.00)
Sales, service, production, transport, laborers	14 (0.01)	141 (0.07)	245 (0.12)	127 (0.06)	57 (0.03)	26 (0.01)	7 (0.00)
Age left school (years)							
≤14	46 (0.02)	343 (0.13)	387 (0.15)	300 (0.11)	151 (0.06)	35 (0.01)	8 (0.00)
≥15	50 (0.02)	395 (0.15)	449 (0.17)	274 (0.10)	135 (0.05)	43 (0.02)	9 (0.00)
Qualifications							
Secondary school	64 (0.03)	404 (0.16)	533 (0.21)	349 (0.14)	164 (0.06)	46 (0.02)	11 (0.00)
Postsecondary, Nontertiary	25 (0.01)	255 (0.10)	218 (0.09)	163 (0.06)	82 (0.03)	22 (0.01)	4 (0.00)
Tertiary	1 (0.00)	65 (0.03)	63 (0.02)	34 (0.01)	20 (0.01)	7 (0.00)	1 (0.00)
Country of birth							
Born in Australia	62 (0.02)	522 (0.20)	650 (0.25)	426 (0.16)	197 (0.07)	54 (0.02)	14 (0.01)
Not born in Australia	34 (0.01)	218 (0.08)	189 (0.07)	150 (0.06)	89 (0.3)	26 (0.01)	4 (0.00)
Language first spoken							
English	93 (0.04)	685 (0.26)	801 (0.31)	550 (0.21)	280 (0.11)	76 (0.03)	18 (0.01)
Other	3 (0.00)	52 (0.02)	34 (0.01)	24 (0.01)	6 (0.00)	3 (0.00)	0 (0.00)
MMSE	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*
	28.5 (2.17)	28.1 (1.99)	27.2 (2.75)	26.6 (3.08)	25.8 (3.09)	25.1 (3.43)	22.1 (5.24)

Note. $N = 2{,}636$. MMSE: Mini-Mental State Examination.

where concurrent Schonell data were unavailable; this preserves missing data patterns that are conditional on factors other than survey design, such as respondent characteristics. Conditional ordinary least squares mean imputation is attractive because it is simple, produces a single estimate, is based on existing relations in the data, and provides more accurate estimates than unconditional mean imputation or follow-up NART scores carried backward. Stochastic regression or multiple imputation, however, remain preferable as they provide more robust imputation procedures with closer approximations of the conditional distributions and, in the case of multiple imputation, account for the uncertainty of predicted values (Graham, 2009; Little & Rubin, 2002; Schafer & Graham, 2002). For this reason, results of the conditional mean imputation procedure will be compared with multiple imputation of five datasets.

Criteria for assessing imputed values

Criteria for the most reliable estimates were the fit of distribution properties, the shared variance between predicted and observed NART scores, and the time-lagged associations between the NART and Schonell within the SOPS sample.

Generation of norms

NART norms were reported for subgroups stratified by 5-year birth cohorts, sex, and a binary measure of age left school using harmonized baseline data from ALSA, CLS, and SOPS. Analysis of covariance (ANCOVA) was used to test for study effects and to investigate the relation between the NART and sociodemographic variables.

Evaluation of age differences and age changes

Mixed models (Singer & Willett, 2003) were used to estimate annualized within-person change in NART scores to provide a comparison with age differences in NART performance based on cross-sectional data. Unadjusted and adjusted (controlling for study, sex, age left school, and total MMSE) estimates were computed using STATA 10 (2007).

RESULTS

Baseline NART prediction for SOPS

At follow-up for SOPS, NART scores ($M = 25.96$, $SD = 9.98$) were lower than the Schonell scores ($M = 35.47$, $SD = 10.7$). The Schonell scores were not normally distributed and were negatively skewed at both waves (baseline: –1.08; follow-up: –1.01). To correct for the negative skew, a square root transformation was applied. After the transformation, Schonell scores had a skew of 0.13 ($SE = 0.11$) at baseline and 0.36 ($SE = 0.13$) at follow-up.

No sex differences in Schonell scores were observed at baseline (mean difference = 1.4; $p = .14$). At follow-up, however, males performed better tban females on both the NART (mean difference = 3.3, $p < .001$) and Schonell (mean difference = 2.8, $p = .02$). This difference in scores was thought to reflect the greater propensity of lower functioning males to drop out of the study due to morbidity or mortality-related factors. We therefore investigated sociodemographic differentials for mortality and attrition. Chi square test revealed no sex differences in attrition not associated with mortality ($\chi^2 = 0.01$, $p = .94$); however, males were more likely to die between baseline and first follow-up ($\chi^2 = 13.9, p < .001$). We also regressed a binary indicator of mortality prior to the first follow-up wave on age, sex, MMSE, and age left school. Participants who died between baseline and first follow-up were more likely to be older (odds ratio, $OR = 1.1$, $SE = .03$, $p < .001$), male ($OR = 2.9$, $SE = .80$, $p < .001$) and score below 23 on the MMSE ($OR = 1.8$, $SE = .57$, $p = .038$). Age left school was not associated with mortality between baseline and first follow-up ($OR = 1.2$, $SE = .30, p = .41$).

Relation between the NART and Schonell

The Schonell scores were strongly associated with NART scores ($r = .913$, $p < .001$; Figure 1). The scatter plot in Figure 1 also revealed ceiling effects for the Schonell, particularly for individuals with more years of schooling. Table 2 shows the bivariate correlations between the NART and candidate covariates for the predictor model. Due to the strong association between the Schonell and the NART, the effects of age, sex, and MMSE on NART scores were not significant and were dropped from the final imputation model. It should not be inferred that these covariates are not predictive of NART performance; rather, this is an indication of the functional equivalence between the NART and Schonell. The exclusion of age, sex, and MMSE from the imputation model is desirable as future analyses that model associations between these covariates and verbal abilities indexed by the NART will not produce inflated covariances. At follow-up, the main effects of Schonell scores (linear and quadratic components) and age left school accounted for 87% of variability in NART scores.

SOPS baseline NART prediction from Schonell scores

Only follow-up NART scores, time-varying Schonell scores, and a continuous measure of age left school were retained in the final imputation model used to predict baseline NART scores for SOPS participants. Eight imputation models were compared, and all explained between 83.9% and 86.8% of variability in NART scores. Models that included age left school imputed baseline NART scores for 465 participants and imputed follow-up NART scores for 3 participants. Models that excluded age left school from the model imputed baseline NART scores for 476 participants and imputed

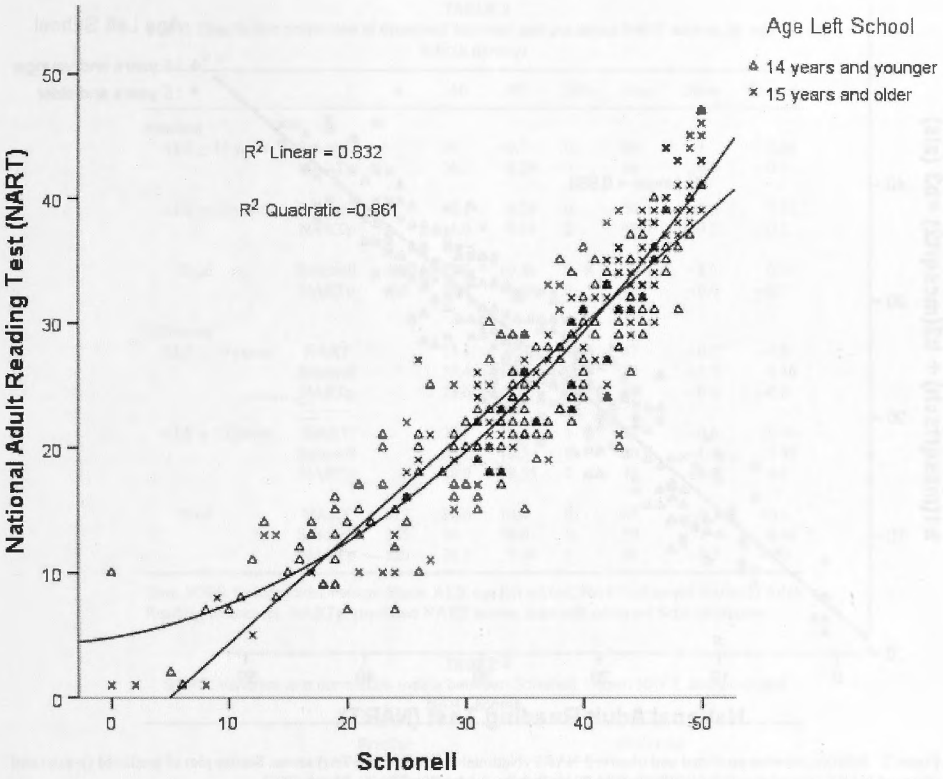

Figure 1. Relation between NART (National Adult Reading Test) and Schonell scores. Scatter plot of raw NART and Schonell scores by age left school for SOPS (Sydney Older Persons Study) at Wave 2 ($r = .91, p < .001$). The relationship is best modeled with both a linear and a quadratic component ($r = .93, p < .001$).

follow-up NART scores for 8 participants. Models that did not correct for the skew in Schonell scores systematically underestimated NART scores for early school leavers and also appeared to be a poor fit of the distribution properties for the NART. Models that excluded the quadratic effect overestimated ability levels of individuals with low NART scores at follow-up. The final model was selected as it showed the smallest standard error ($SE = 3.65$) and showed the closest match to the distribution of observed NART scores at follow-up (Figure 2, Table 3).

TABLE 2
Pairwise bivariate correlations between the NART, Schonell, and covariates at follow-up for SOPS

	NART	*Schonell*	*√Schonell*	*MMSE*	ALS_{cont}
NART	1				
$Schonell_{raw}$	.91**	1			
$√Schonell_{trans}$	−.93**	−.97**	1		
MMSE	.52**	.56**	−.52**	1	
ALS_{cont}	.31**	.26**	−.29**	.03	1
Age	−.22**	−.19**	.19**	−.22**	−.09

Note. $N = 345$. MMSE: Mini-Mental State Examination. NART: National Adult Reading Test. SOPS: Sydney Older Persons Study. ALS_{cont}: age left school, continuous measure; $Schonell_{raw}$: raw Schonell scores; $Schonell_{trans}$: square-root transformed Schonell scores.
**$p < .01$, two-tailed.

The predicted NART scores were not rounded to the nearest whole number and were imputed by the unstandardized regression equation:

$$NART_{pred} = (20.651) + (-6.225) * (Schonell_{trans}) + (-.225) * (Schonell^2_{trans}) + (.399) * (als),$$

where $Schonell_{trans}$ is the mean centred transformed Schonell score, $Schonell_{trans}{}^2$ is the quadratic effect for the mean centered transformed Schonell score, and *als* is age left school. There was an average decline of 2.39

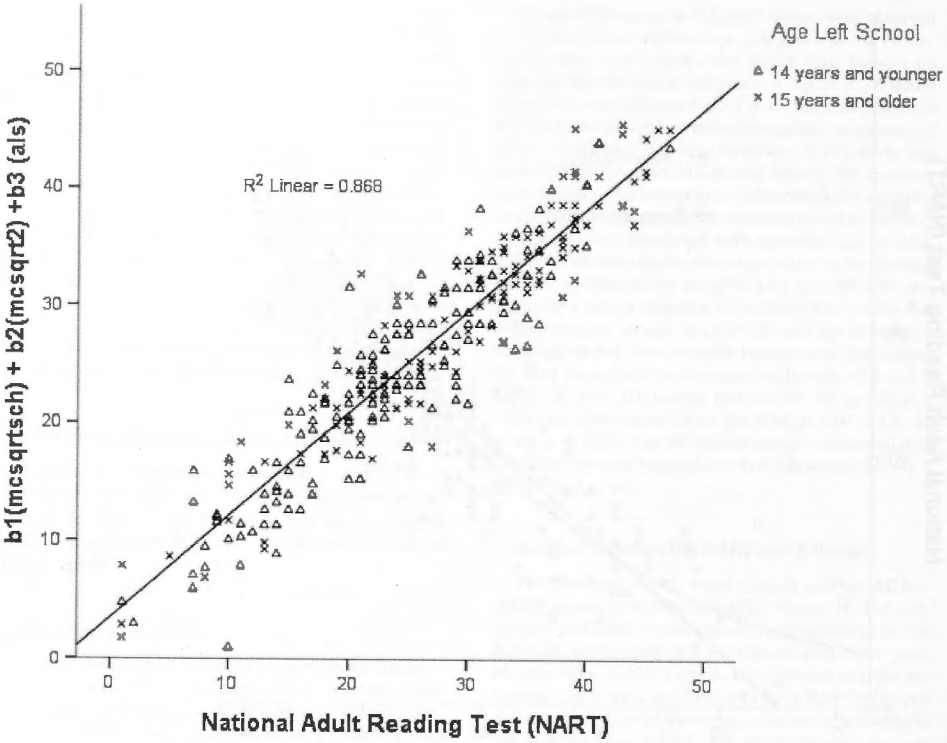

Figure 2. Relation between predicted and observed NART (National Adult Reading Test) scores. Scatter plot of predicted (*y*-axis) and observed NART (*x*-axis) scores for SOPS (Sydney Older Persons Study) Wave 2 ($r = .93, p < .001$).

points ($SD = 4.81$) in NART performance between baseline and follow-up ($M_{years} = 2.9$, $SD = 0.3$), compared to an average decline of 2.61 points ($SD = 4.77$) on the Schonell. The correlation between the NART and Schonell difference scores was $r = .61$ ($p < .001$), indicating a moderate to strong association in change over two waves between these two measures.

The variances and correlations between predicted and observed NART and Schonell scores across both waves are presented in Table 4. Correlations ranged between .85 and .98. No significant differences were found between predicted baseline NART scores estimated by imputation ($M = 28.22$, $SE = 0.44$) or multiple imputation ($M = 28.11$, $SE = 0.53$), $t(10) = 0.363$, $p = .725$; $r = .93$, $p < .001$. It is evident that the final mean imputation model underestimated variability, as predicted NART scores had a standard deviation of 9.38 at follow-up, whereas the actual NART standard deviation was 10.4. Further, the mean standard error was larger for estimates derived from multiple imputation. This was not unexpected and is a common problem of conditional mean imputation as predicted values do not deviate from the regression line (Graham, 2009). Baseline NART norms for the SOPS sample are based on the values imputed by conditional mean imputation.

NART norms

Normative data

Only the CLS provided item-level data that could be used to investigate the psychometric properties of the NART. Reliability analysis for this sample revealed that Cronbach's alpha was comparable across all age groups and ranged from .85 to .93 (Table 5). This is generally consistent with previous findings. For example Uttl (2002) reported alphas of .92, .94, and .93 for broad age cohorts (young, 18–39 years; middle-aged, 40–59 years; and older adults, 60–91 years, respectively). The relatively lower alpha of .85 reported for this sample was for the oldest age group (95+), which had a small sample size ($n = 18$) and comprised an age cohort not captured in the Uttl (2002) sample. Luszcz et al. (1997) reported test–retest reliability of .83 across all ages ranging from 65 to 103 years. Test–retest reliability is considered a more

TABLE 3
SOPS: Distribution properties of observed Schonell and predicted NART scores, by age left school (binary)

		n	*M*	*SD*	*Min*	*Max*	*Skew*	*Kurtosis*
Baseline								
ALS ≤ 14 years	Schonell		36	10.7	0	50	−1	0.56
	NARTp		26.1	9.29	0	44	−0.3	−0.3
ALS ≥ 15 years	Schonell		40.1	9.29	0	50	−1.3	1.73
	NARTp		31.0	9.04	2	48	−0.5	−0.2
Total	Schonell	465	37.8	10.3	0	50	−1.1	0.92
	NARTp	465	28.2	9.49	0	48	−0.4	−0.3
Follow-up								
ALS ≤ 14 years	NART		23.4	8.84	1	47	−0.1	−0.4
	Schonell		33.4	10.5	0	50	−0.7	0.16
	NARTp		23.6	8.61	1	44	−0.1	−0.2
ALS ≥ 15 years	NART		29.1	10.4	1	47	−0.6	−0
	Schonell		38.1	10.5	0	50	−1.4	1.99
	NARTp		28.9	9.33	2	46	−0.6	0.1
Total	NART	317	25.6	10.4	0	47	−0.3	−0.4
	Schonell	325	36	10.6	0	50	−1	0.66
	NARTp	320	26.5	9.38	1	48	−0.2	−0.3

Note. SOPS: Sydney Older Persons Study. ALS: age left school; NART: observed National Adult Reading Test scores; NARTp: predicted NART scores; Schonell: observed Schonell scores.

TABLE 4
SOPS: Variance and correlation matrix between Schonell, known NART, and predicted NART scores

	Baseline		*Follow-up*		
	Schonell	*NART predicted*	*Schonell*	*NART original*	*NART predicted*
Baseline					
Schonell	104.79				
NART predicted	.98**	89.95			
Wave 2					
Schonell	.88**	.88**	116.90		
NART original	.85**	.88**	.913**	107.81	
NART predicted	.87**	.89**	.98**	.93**	87.89

Note. NART: National Adult Reading Test. SOPS: Sydney Older Persons Study.
**$p < .01$, two-tailed.

conservative reliability estimate relative to measures of internal consistency such as Cronbach's alpha.

Table 5 shows the normative data at baseline for the NART in five-year age groups by sex and age left school. ANCOVA revealed that NART performance was related to age left school and qualifications attained. Individuals who left school before the age of 15 years ($M = 25.46$, $SD = 9.22$, $n = 1{,}270$) performed worse than participants who left school at ages 15 years or older ($M = 31.74$, $SD = 9.69$, $n = 1{,}355$; $p < .001$). Individuals with tertiary qualifications ($M = 38.87$, $SD = 8.14$, $n = 191$) performed better than individuals with postsecondary nontertiary qualifications ($M = 30.23$, $SD = 8.14$, $n = 769$) who performed better than individuals with secondary school qualifications only ($M = 26.83$, $SD = 9.69$, $n = 1{,}571$). A negative association was also present between age and NART scores: Every 10 years lived resulted in a predicted difference of −1 point in NART score ($p < .001$). No sex effects were found ($p = .47$) after adjusting for age and education.

Care must be taken when interpreting age differences in NART performance as this finding is based on cross-sectional analysis, which cannot distinguish between age and cohort effects, nor permit inference concerning within-person change. To examine this further, subsequent longitudinal analysis of the pooled sample tested

TABLE 5

Normative baseline NART scores from the pooled DYNOPTA dataset by 5-year age group, sex, and age left school

			Age group						
			<70 (n = 96)	*70–74 (n = 740)*	*75–79 (n = 839)*	*80–84 (n = 577)*	*85–89 (n = 286)*	*90–94 (n = 80)*	*≥95 (n = 18)*
		Alpha		.92	.93	.92	.92	.91	.85
Whole sample		*n*	96	740	839	577	286	80	18
		M (SD)	27.3 (8.6)	29.7 (9.92)	28.9 (10.19)	28.2 (9.82)	27.1 (9.73)	27.3 (10.84)	25.5 (9.63)
		Range	4–45	0–50	0–49	0–50	0–50	0–50	9–40
Males	ALS ≤ 14 years	*n*	5	180	217	176	89	17	2
		M (SD)	20.6 (10.31)	27.0 (9.52)	25.6 (10.1)	26.1 (8.9)	24.2 (9.47)	21.2 (11.34)	24.0 (11.31)
		Range	9–32	0–46	0–44	2–50	4–50	0–36	16–32
	ALS ≥ 15 years	*n*	5	207	234	122	72	16	2
		M (SD)	25.4 (7.23)	33.4 (9.76)	32.3 (9.74)	32.0 (10.19)	29.9 (9.52)	29.8 (8.56)	33.5 (6.04)
		Range	18–35	0–50	2–48	0–48	10–47	13–50	29–38
	Total	*M (SD)*	23 (8.77)	30.4 (10.16)	29.1 (10.46)	28.5 (9.87)	26.8 (9.88)	25.2 (10.77)	28.7 (9.2)
Females	ALS ≤ 14 years	*n*	41	163	170	124	62	18	6
		M (SD)	23.6 (7.71)	25.7 (8.53)	25.3 (9)	24.8 (8.57)	24.9 (9.59)	24.4 (7.41)	25.0 (9.61)
		Range	4–38	1–45	0–44	1–50	0–42	9–40	14–40
	ALS ≥ 15 years	*n*	45	188	215	152	63	27	7
		M (SD)	31.6 (7.36)	32.0 (9.4)	31.5 (9.78)	30.7 (9.9)	30.1 (8.86)	31.4 (11.79)	26.1 (10.04)
		Range	18–45	5–49	6–49	6–50	11–49	1–47	9–39
	Total	*M (SD)*	27.8 (8.49)	29 (9.61)	28.8 (9.89)	27.9 (9.78)	27.5 (9.56)	28.9 (10.75)	24.6 (9.88)

Note. Cronbach's alpha based on the Canberra sample only. Test–retest reliability has previously been reported in the Adelaide sample across all age groups to be .83 (Luszcz et al., 1997). NART: National Adult Reading Test. DYNOPTA: Dynamic Analyses to Optimise Ageing. ALS: age left school.

for age trajectories of intraindividual change in NART score. Participants were assessed on the NART on up to four occasions ($M = 1.9$), and the average time interval between waves was 2.2 years ($SD = 0.7$; range 0.09–5.9). Unadjusted linear mixed models revealed an average decline in NART performance of 1.4 words correctly pronounced over 10 years ($SE = 0.17$, $p < .001$). However, after adjusting for study, sex, age left school (binary), and total MMSE score, the average rate of decline in NART attenuated to 0.4 words correctly pronounced over 10 years ($SE = 0.17$, $p = .02$).

Study differences

Differences in baseline NART performance across studies were observed: The Canberra sample ($M = 30.5$, $SD = 11.7$) scored higher than both Sydney ($M = 28.2$, $SD = 9.58$, $p < .001$) and Adelaide ($M = 27.52$, $SD = 8.48$, $p < .001$) samples. After adjusting for age, sex, age left school, qualifications attained, language first spoken, and MMSE, study differences were no longer present between the estimated marginal means for the Sydney and Canberra samples ($p = .75$), although lower performance for Adelaide participants remained ($p < .001$).

DISCUSSION

This study investigated the functional equivalence of two irregular word pronunciation tests and found that the Schonell and NART have a strong linear relation, with a slight curvilinear component. These findings indicate that NART scores can be estimated based on the Schonell and age left school. Given the strength of the relation between the tests, little benefit is gained from administering each measure concurrently aside from the opportunity to investigate their functional equivalence.

Imputation of baseline NART scores for SOPS participants ensures that NART norms reported for this population are not biased by participative effects, attrition, or mortality. Although verbal abilities indexed by the NART are not expected to decline with age, NART performance may decline in relation to disease or mortality processes (White & Cunningham, 1988). Thus, baseline estimates also lay the groundwork for future investigations into individual differences in change of NART scores. Salthouse (2009) has recently argued that the true extent of age-related declines in cognition will be largely obscured in longitudinal studies that do not account for learning effects, to which verbal tasks are highly susceptible. The use of alternate measures is one approach to minimizing these participative effects. This paper therefore joins a growing realization that identical measures need not be applied across all measurement occasions of longitudinal studies. Longitudinal analyses can be applied to changing scales of functionally equivalent measures (McArdle et al., 2009) and benefit from planned missingness designs (Graham, Taylor, & Cumsille, 2001). Although in this instance the opportunity to investigate the functional equivalence of two measures of verbal abilities was largely serendipitous, researchers involved in the design of future longitudinal studies may wish to consider incorporating alternate forms of functionally equivalent measures across waves.

The second aim of this study was to combine three large population-based studies to report normative NART data with greater precision and representation of a broader national population. This is a key strength of the study as historically normative NART data have been based on relatively small samples of recruited volunteers or from community groups via advertisements and are not drawn from a random sample of the general population.

Our investigations confirmed that educational attainment is related to NART scores: Early school leavers systematically performed worse than participants who completed secondary schooling. Likewise, those failing to obtain a postsecondary schooling qualification were more likely to incorrectly pronounce irregular words than individuals who did obtain further postsecondary and tertiary qualifications. Sex did not account for any variability in NART scores after adjusting for sociodemographics, which is consistent with previous findings. Contrary to reported norms for the North American Adult Reading test (NAART), which show improved NART performance in older adults (Strauss et al., 2006; Uttl, 2002), older adults were more likely to perform worse than their younger counterparts in this study. This difference, however, is most likely due to the greater age range (18–91 years, $n = 351$) of the sample available to Uttl than that in this study (65–103 years, $n = 2{,}636$). Perhaps of greater interest is the gradual decrease in NART performance with age. Age-related rates of decline remained after adjusting for basic sociodemographics and MMSE score, though they were greatly reduced. We did not adjust for health, mortality, or other dementia risk factors, which may further account for age-related declines in verbal abilities, as this was beyond the scope of this study. However, as previously mentioned, the possibility that true decline rates were masked by practice effects cannot be discounted. Thus it is important to note that despite the strongly supported notion of age stability for putative crystallized abilities in late adulthood (Baltes, Staudinger, & Lindenberger, 1999), subtle declines in verbal abilities, as indexed by the NART, may be expected in the general population.

The issue of cohort differences across studies is intriguing as it does not arise in typical reports of normative data that rely on single study samples. Even after adjusting for sociodemographic and health variables, the Adelaide sample on average scored lower than the Sydney and Canberra samples. This could be due to respondent characteristics not accounted for in the analyses, or study differences in administration protocols. Regardless of the underling cause of these study differences in NART performance, this finding demonstrates a further advantage of analyzing harmonized and pooled data. The representativeness of the broader national population is enhanced

while study-specific sample biases, which would otherwise be undetected, are minimized.

Limitations

Although imputation by ordinary least squares regression underestimated the variability in baseline NART scores for SOPS participants, the comparable distributions and strong association between the Schonell and NART indicate that this was of little consequence. In this context, little benefit was to be gained by predicting NART scores via more computationally and analytically complex missing data methods such as multiple imputation.

Responses to individual items were not available for the NART and Schonell in two of the three studies, which only coded summed total scores. Absence of individual item responses prevents reliability analyses, investigation of item characteristics, and the use of more robust techniques of harmonization such as those orientated by item response theory. This is an important reminder of the importance of having access to raw item-level data.

A possible shortcoming of this study is that these NART norms do not include participants from a nonurban population. In addition, we do not report NART norms for adults under the age of 65 years and acknowledge the small cell counts for adults aged over 95 years.

In summary, this study successfully demonstrates the benefits afforded by the harmonization of functionally equivalent measures and reports NART norms for adults aged 65 years and older that have increased precision and representativeness than those previously available. These data are of considerable clinical importance and will be of significant value to clinicians and researchers who work with older cohorts and apply the NART to obtain a proxy of premorbid verbal IQ and verbal functioning.

Original manuscript received 7 June 2010
Revised manuscript accepted 17 September 2010
First published online day month year

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J Aging Health published online 28 December 2011
DOI: 10.1177/0898264311425088

The online version of this article can be found at:
http://jah.sagepub.com/content/early/2011/12/21/0898264311425088

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Journal of Aging and Health
XX(X) 1–20

DOI: 10.1177/0898264311425088
http://jah.sagepub.com
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Evaluating a Dichotomized Measure of Self-Reported Hearing Loss Against Gold Standard Audiometry: Prevalence Estimates and Age Bias in a Pooled National Data set

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Abstract

Objectives: To evaluate a harmonized binary measure of self-reported hearing loss against gold standard audiometry in an older adult population. **Method:** Seven nationally representative population-based studies were harmonized and pooled (n = 23,001). Self-report items were recoded into a dichotomous format. Audiometric hearing loss was defined by averaged pure-tone thresholds greater than 25-decibel hearing level in the better ear. We compared age and sex stratified prevalence rates of hearing loss estimated by self-report and audiometric measures. **Results:** Overall, 56% of men and 43% of women had audiometric hearing loss. There were moderate associations between self-reported and audiometric hearing loss. However, prevalence based on self-report was overestimated for adults aged below 70

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years and underestimated for adults aged above 75. **Discussion:** Self-report of hearing loss is insensitive to age effects and does not provide a reliable basis for estimating prevalence of age-related hearing loss, although may indicate perceived hearing disability.

Keywords

age-related hearing loss, presbycusis, harmonization, data pooling, the Australian Longitudinal Study of Ageing (ALSA), the Australian Longitudinal Study of Women's Health (ALSWH), the Blue Mountains Eye Study (BMES), the Canberra Longitudinal Study (CLS), the Melbourne Longitudinal Studies on Healthy Ageing Program (MELSHA), the Path Through Life Project (PATH), the Sydney Older Persons Study (SOPS)

Hearing loss is among the most prevalent chronic age-related health conditions. It occurs in up to 90% of American adults aged above 80 (Cruickshanks et al., 2003), and is estimated to currently affect 75% of Australians aged above 70 years (Access Economics, 2006). Overall, Australian prevalence rates are projected to increase with population aging. In 2005, the financial cost and burden of disease arising from hearing loss was estimated to be more than AU$22 billion in Australia (Access Economics, 2006) and in excess of €213 billion Europe (Shield, 2006). The lifetime cost of severe to profound hearing loss in the United States has been estimated to be US$297,000 per person (Mohr et al., 2000). These estimates are also expected to increase with rising prevalence. Age-related hearing loss has been linked with reduced quality of life (Chia et al., 2007; Hogan, O'Loughlin, Miller, & Kendig, 2009), poor mental health (Gopinath, 2009b; Kramer, Kapteyn, Kuik, & Deeg, 2002), diabetes (Mitchell et al., 2009), smoking (Gopinath et al., 2010), cognitive impairment (Tay et al., 2006), reduced social participation, increased use of community support services (Schneider et al., 2010), and increased risk of mortality (Karpa et al., 2010). Despite its impact on health and well-being, age-related hearing loss is known to be both underrecognized and undertreated (Reuben, Walsh, Moore, Damesyn, & Greendale, 1998). Recent reviews have found that globally only a small number of surveys were suitable for estimating hearing impairment in the general population (Pascolini & Smith, 2009). There are currently sparse national data on hearing impairment in older people, in Australia and elsewhere, with recent investigations of risk factors for incidence of age-related hearing loss being underpowered (Gopinath et al., 2010; Gopinath, Schneider, Rochtchina, Leeder, & Mitchell, 2009a; Mitchell et al., 2009).

The gold standard method for measuring hearing loss is pure-tone audiometry. The World Health Organisation (WHO) defines mild hearing impairment as unaided pure-tone audiometric hearing thresholds (PTA) greater than 25-decibel hearing level (dB HL) in the better ear, averaged across the tone frequencies of 0.5, 1, 2, 4 kHz. Hearing thresholds greater than 40dB HL are described by WHO as disabling (World Health Organization, 1999). Definitions of moderate and severe hearing loss are slightly different in the United States. Because the costs and logistics involved in conducting audiometry assessments are prohibitive for many epidemiological surveys, self-report measures are often used instead. These self-reported measures of hearing loss have previously been thought to be reliable and predictive of measured hearing loss while also providing an ecologically valid measure of perceived hearing difficulties (Johnson, 2010; Nondahl et al., 1998).

Contrary to the belief that self-reported hearing items are strongly associated with measured hearing loss, evidence is equivocal. Self-reported difficulty of hearing a conversation in a quiet room has been shown to have serious misclassification problems of audiometric hearing loss and has been argued to be inappropriate for providing prevalence estimates of hearing impairment over a broad age sample (range 15 to 71+; Wilson et al., 1999). Response propensity may also be influenced by individual differences, as one study has shown that self-reported hearing loss is more strongly associated with personality than measured hearing loss (Cox, Alexander, & Gray, 2007). In contrast, other studies (Nondahl et al., 1998; Sindhusake et al., 2001) have reported that a more general question asking "Do you feel you have hearing loss?" with binary response is a more reliable self-report measure for assessing age-related hearing loss in adults aged between 45 and 100, with better predictive properties compared to other self-report scales such as the Hearing Handicap for the Elderly (HHIE; Ventry & Weinstein, 1982), hearing aid use, and hearing ratings on a 5 point Likert-type scale, although these comparisons failed to consider how age moderates the association between self-report and audiometric hearing measures. Although responses are generally coded on a 3- to 5-point Likert-type scale, it is not uncommon to recode responses into a dichotomous variable format, whereby any degree of reported hearing difficulty (e.g., "a little trouble," "a lot of trouble, " "deaf") is taken to be indicative of hearing loss (Caban, Lee, Gomez-Marin, Lam, & Zheng, 2005).

Given the lack of large-scale data sets including information on hearing loss, much benefit can be gained from combining information from the available data sets that include items on hearing loss. This data-pooling approach requires harmonizing and validating measures of self-reported hearing loss so they are comparable across surveys. In this study, we report on a method

of dichotomizing self-reported hearing loss items in a pooled data set and evaluate the reliability and utility of such harmonized self-reported hearing loss in comparison to the gold standard measure of pure-tone audiometric thresholds. The aims of the present study were, first, to compare prevalence estimates of hearing loss between those based on dichotomized self-report and standardized audiometric measurements. Second, this study aimed to evaluate the utility of a harmonized binary measure of self-reported hearing loss. The findings will enable future research into prevalence and incidence of hearing impairment, its risk factors and outcomes, in a nationally representative pooled data set.

Method

Sample

Data were drawn from the Dynamic Analyses to Optimise Ageing Project (DYNOPTA) which has harmonized and pooled nine Australian longitudinal studies of aging (baseline $n = 50,652$). A comprehensive description of the study design has been reported previously (Anstey et al., 2010). Baseline details of the contributing studies included in this article are shown in Table 1 and comprise the Australian Longitudinal Study of Ageing (ALSA; Luszcz et al., 2007), the Australian Longitudinal Study of Women's Health (ALSWH; Lee et al., 2005), the Blue Mountains Eye Study (BMES; Mitchell et al., 2009), the Canberra Longitudinal Study (CLS; Christensen et al., 2004), the Melbourne Longitudinal Studies on Healthy Ageing Program (MELSHA; Browning & Kendig, 2010), the Path Through Life Study (PATH; Anstey et al., 2011) and the Sydney Older Persons Study (SOPS; Piguet et al., 2003). Baseline waves were collected between 1992 and 2001. Table 2 shows the pooled sample profile reported in this study ($n = 23,001$; 22% men). At baseline, the mean age was 71.6 years ($SD = 6.2$, range = 55 to 103), 9,190 (40%) participants left secondary school before the age of 15 years and 1,759 (8%) participants were tertiary qualified. The preponderance of women aged between 70 and 75 years reflects the inclusion of the ALSWH old cohort ($n = 12,432$).

Measures

Measured hearing loss was assessed by uncorrected pure-tone air-conduction audiometric thresholds (PTA) in waves 1, 3, 6, and 7 of ALSA and waves 2 and 3 of BMES. We defined hearing impairment as PTA greater than 25 dB HL in the better ear averaged across air-conduction tone frequencies of 0.5,

Table 1. Contributing Study Baseline Information

Study	Location	Baseline year	*n*	Age range
Australian Longitudinal Study of Ageing (ALSA)	Adelaide, SA	1992	2087	65-103
Australian Longitudinal Study of Women's health (ALSWH)	Australia (national)	1996	13706	45-51
Blue Mountains Eye Study (BMES)	Blue Mountains, NSW	1997-00	2334	50-98
Canberra Longitudinal Study (CLS)	Canberra, ACT Queanbeyan, NSW	1990-91	1134	70-103
Melbourne Longitudinal Study Healthy Ageing (MELSHA)	Melbourne, Vic	1994	1000	65-94
Personality and Total Health through life (PATH)	Canberra, ACT Queanbeyan, NSW	2005-06	2222	64-70
Sydney Older Person's Study (SOPS)	Sydney, NSW	1991-93	630	75-97

1, 2, and 4 kHz ($PTA_{0.5,1,2,4\ kHz}$). Levels of hearing impairment were defined as no hearing loss ($PTA_{0.5,1,2,4\ kHz} < 25$ dB), mild hearing loss ($PTA_{0.5,1,2,4\ kHz}$: 25-40 dB), moderate hearing loss ($PTA_{0.5,1,2,4\ kHz}$: 41-60 dB), and severe hearing loss ($PTA_{0.5,1,2,4\ kHz} > 60$ dB).

Self-reported hearing loss was obtained by ALSA (Waves 1, 3, 6, 7), ALSWH (Wave 1), BMES (Waves 1, 2, 3), CLS (Waves 1, 2, 3, 4), MELSHA (Waves 1, 10), PATH (Wave 2) and SOPS (Waves 1, 2, 4, 5). Original item wording and response formats varied between studies and are presented in Table 3. A harmonized measure of self-reported hearing loss comprising of two levels was created ("no self-reported hearing loss," and "any self-reported hearing loss"), whereby any degree of reported hearing losses, hearing difficulty, or hearing problems were recoded to reflect self-reported hearing loss.

Table 2. DYNOPTA Sample Profile at Baseline, Demographics (Row %) by 5-Year Age Groups (N = 23,001)

		55-59	60-64	65-69	70-74	75-79	80-84	85+
	n	%	%	%	%	%	%	%
Sex								
Men	5061	4.5	28.8	12.6	17.1	17.4	11.4	8.1
Women	17940	1.7	8.0	4.6	71.1	8.2	3.5	2.9
Age left school								
Left school age 14 or younger	9190	0.7	4.8	5.3	64.0	12.4	7.2	5.6
Left school age 15 or older	13215	3.5	18.5	7.4	55.5	8.7	3.9	2.5
Highest qualification								
Secondary school	14708	1.3	5.4	4.3	70.8	10.2	4.7	3.3
Nontertiary	5204	4.8	24.8	11.9	37.2	10.6	6.6	4.1
Tertiary	1759	2.9	42.3	9.2	31.3	7.2	4.0	3.1
Career occupation								
Managers and professionals	3933	1.7	26.1	7.5	43.9	10.2	6.1	4.5
Clerical and associate professional	5349	2.8	12.4	6.3	64.0	8.4	3.7	2.4
Tradespersons	1466	2.9	11.0	8.8	44.5	15.7	10.1	7.1
Sales, service, production, transport and laborers	5640	2.6	15.2	6.8	56.0	10.9	4.7	3.7

Note. Pooled baseline sample from ALSA, ALSWH old cohort, BMES, CLS, MELSHA, PATH.

Age left school was coded as a binary variable indicating school leaving age being 15 years or older.

Analyses

Self-reported hearing measures were obtained concurrently with audiometric assessments in ALSA Waves 1, 3, 6, and 7 and BMES waves 2 and 3. Evaluation of self-reported hearing measures was made by investigating their association with audiometry within these samples and waves. First, the sample was classified according to the categories of the self-report measure, then the means and standard deviations for $PTA_{0.5,1,2,4\ kHz}$ were calculated to

Table 3. Self-Reported Hearing Items Harmonized in DYNOPTA

		Item	Response			
Study	Wave	Harmonized self-reported hearing	Normal hearing	Hearing impairment		
ALSA (Luszcz et al., 2007)	1	Do you have any difficulty with your hearing?[a]	No	Yes		
	1	If yes, how much difficulty do you have with your hearing?[a]		Slight	Moderate	Severe
	3, 6, 7	How much difficulty, if any, do you have with your hearing (even if you are wearing your hearing aid)?[a]	None	Slight difficulty	Moderate difficulty	Great difficulty
ALSWHold (Lee et al., 2005)	1	In the past 12 months have you had any hearing problems	Never	Rarely	Sometimes	Often
BMES (Mitchell et al., 2009)	1	Have you ever had a problem with your hearing	No	Yes		
	1	Assessment of hearing problem		Mild	Moderate	Severe
	2, 3	Do you feel you have hearing loss[a]	No	Yes		
	3	Do you have difficulties with your hearing?[a]	No	Sometimes	Yes	
CLS (Christensen et al., 2004)	1, 2, 3, 4	Would you say your hearing (with a hearing aid) is generally good, fair or poor?	good	fair	poor	
MELSHA (Browning & Kendig, 2010)		Is your hearing (with your hearing aid)	Excellent or Good	Fair	Poor	
PATH (Anstey et al., 2011)	2	How you would rate your hearing on the following scale?	Adequate for all purposes	Slight inconvenience	Definite inconvenience	Definite handicap
SOPS (Piguet et al., 2003)	1, 2	Do you have any loss of hearing	No	Yes		

a. Validated against gold standard of PTA (pure tone thresholds) averaged over 0.5, 1, 2, 4 kHz in the better ear.

enable calibration between subjective and objective measures. Second, polychoric correlations between the defined levels of impaired hearing thresholds (none, mild, moderate, severe) in the better ear and self-reported hearing were estimated using Mplus v5 (Muthen & Muthen, 2007). Finally, the discriminative properties of dichotomized measures were evaluated using receiver operator characteristic (ROC) curves, sensitivity, and specificity.

Baseline prevalence estimates for hearing loss were stratified by 5-year age groups and sex. Prevalence were estimated for pooled audiometric data from ALSA Wave 1 and BMES Wave 2, pooled dichotomized self-report data from ALSA Wave 1 and BMES Wave 2, and pooled dichotomized self-report data from all contributing studies. To examine how well self-reported hearing data could replicate findings based on analysis of $PTA_{0.5,1,2,4\ kHz}$ in multivariate analyses, odds ratios (OR) were estimated from a logistic regression model testing the effects of age, sex, and age left school on audiometric measures of hearing loss and on dichotomized self-reported hearing loss. Results were compared between all studies, using Wave 1 from the ALSA, ALSWH, CLS, MELSHA, and SOPS samples, and Wave 2 from the BMES and PATH samples.

Results

Comparison of Self-Report and Audiometric Hearing Loss

The broader age range and inclusion of participants younger than 65 years meant that the overall average hearing thresholds were lower in the BMES sample (Mean [*M*] = 24.9 dB, Standard Deviation [*SD*] = 14.7) compared to the ALSA sample (*M* = 35.4 dB, *SD* = 16.1). Table 4 compares how well four dichotomized self-report items predicted hearing loss as measured by $PTA_{0.5,1,2,4kHz}$ within ALSA and BMES. Three of the self-report items were moderately associated with audiometric hearing loss. Their polychoric correlations ranged between 0.56 and 0.69, and area under the curve (AUC) ranged between 0.75 and 0.82. Furthermore, the average $PTA_{0.5,1,2,4kHz}$ was higher for respondents who reported some level of hearing loss or hearing difficulty compared to respondents who reported no hearing loss or no hearing difficulties (ALSA Wave 1: mean difference = 16.5 dB, $t_{(1593)} = 21.8, p < .01$; ALSA Wave 3: mean difference = 13.0 dB, $t_{(1387)} = 17.3, p < .01$; ALSA Wave 6: mean difference = 12.8 dB, $t_{(490)} = 11.5, p < .01$; ALSA Wave 7: mean difference = 11.5dB, $t_{(349)} = 8.4, p < .01$; BMES Wave 2: mean difference = 14.2 dB, $t_{(1919)} = 23.2, p < .01$; BMES Wave 3: mean difference = 12.5 dB, $t_{(1562)} = 18.5, p < .01$). However, one item from Wave 3 in BMES, "do

Table 4. Hearing Thresholds (dB HL) for Levels of Self-Reported Hearing and Their Association

Self-report	*n*	Age range	Mean PTA[b]	*SD*	ρ (se)	AUC (se)	Sens.	Spec.	PPV	NPV
ALSA wave 1 (1992)[a]										
hearing loss	741	65-101	43.0	17.6	.69	.82 (.01)	.62	.85	.90	.51
no hearing loss	854	65-103	26.5	12.4						
ALSA wave 3 (1994)[c]										
hearing loss	722	66-105	41.6	15.1	.56	.76 (.01)	.63	.78	.88	.45
no hearing loss	667	66-100	28.6	12.7						
ALSA wave 6 (2000)[c]										
hearing loss	262	73-101	42.7	13.8	.56	.77 (.02)	.62	.77	.90	.37
no hearing loss	230	72-99	29.8	10.6						
ALSA wave 7 (2003)[c]										
hearing loss	204	75-102	41.8	12.8	.59	.75 (.02)	.67	.77	.92	.38
no hearing loss	147	76-96	30.3	12.0						
BMES wave 2 (1997-2000)[d]										
hearing loss	985	54-98	31.6	16.4	.66	.78 (.01)	.78	.67	.61	.82
no hearing loss	936	50-96	17.4	9.2						
BMES wave 3 (2001-2004)[d]										
hearing loss	1000	59-99	30.1	14.7	.61	.77 (.01)	.85	.53	.59	.81
no hearing loss	564	55-96	17.5	9.2						
BMES wave 3 (2001-2004)[e]										
hearing loss	787	55-99	26.1	14.3	.03	.51 (.02)	.63	.41	.47	.57
no hearing loss	516	55-96	25.8	14.9						

a. Do you have any difficulty with your hearing?

b. PTA: average pure tone thresholds (decibels hearing levels dB HL) from tone frequencies of 0.5, 1, 2, 4 kHz in the better ear.

c. How much difficulty, if any, do you have with your hearing (even if you are wearing your hearing aid)?

d. Do you feel you have hearing loss?

e. Do you have difficulties with your hearing?

ρ : Polychoric Correlations between self-reported hearing and defined levels of hearing impairment (none, mild, moderate, severe) based on $PTA_{0.5,1,2,4\ kHz}$ in the better ear; AUC: Area Under the Curve, Receiver Operator Characteristic of self-reported hearing loss with $PTA_{0.5,1,2,4\ kHz}$ (dB) in the better ear; Sens.: Sensitivity; Spec: Specificity; PPV: Positive predictive value; NPV: Negative predictive value.

you feel you have difficulties with your hearing?" with responses originally coded on a 3-point scale: *yes*, *sometimes*, *no*, showed poor discrimination between impaired and nonimpaired hearing as defined by $PTA_{0.5,1,2,4\ kHz} > 25$ dB HL. When recoded into dichotomous variable format where a response of either *yes* or *sometimes* indicated self-reported hearing loss, there was little difference in hearing thresholds between categories (mean difference = 0.3dB, $t_{(1301)} = 0.4$, $p = .72$; polychoric correlation = .03; AUC = .51, standard error [*SE*] = .02). Subsequent regression analysis revealed that original responses for this question were not associated with $PTA_{0.5,1,2,4\ kHz}$ ($F_{(2, 1300)} = 0.1$, $p = .90$). This self-report item was not used in coding the harmonized self-reported hearing variable.

There were between-study differences in the ratio of positive predictive value (PPV) relative to negative predictive value (NPV). In all ALSA waves, self-reported hearing loss had high PPV (Wave 1 = .90, Wave 3 = .88, Wave 6 = .90, Wave 7 = .92) and comparably lower NPV (Wave 1 = .51, Wave 3 = .45, Wave 6 = .37, Wave 7 = .38). In contrast, both BMES waves had lower PPV (Wave 2 = .61, Wave 3 = .59) and higher NPV (Wave 2 = .82, Wave 3 = .81). The ALSA item had higher specificity than sensitivity at all waves whereas the item used in BMES showed lower specificity than sensitivity at all waves. The difference between studies can largely be explained by their respective age distributions. To demonstrate this we compared PPV and NPV for an older age cohort (aged 70 years and older, $n = 947$) relative to a younger aged cohort (aged 69 years and younger, $n = 974$) in the Wave 2 sample of BMES. In the older age cohort, the PPV of 0.76 was higher than the NPV of 0.67. This was reversed in the younger age cohort where the PPV of 0.41 was smaller than the NPV of 0.93. This could be expected as audiometric hearing loss is less prevalent at younger ages, consequently a negative test result is more likely in the younger cohort whereas the probability of a positive test result increases with age.

Comparison of Prevalence Rates Based on Self-Report and Audiometric Hearing Loss

Figure 1 shows the prevalence hearing loss based on audiometric assessment and self-report. Within the pooled ALSA and BMES sample, overall prevalence rates derived from self-reported hearing loss was 56% (95% CI = [53, 58]) for men and 43% (95% CI = [41, 45]) for women, which only slightly underestimated true overall prevalence estimated from $PTA_{0.5,1,2,4\ kHz}$, of 59% (95% CI = [57, 61]) for men and 46% (95% CI = [44, 48]) for women. However, the age gradient for self-report items was not as steep as the age

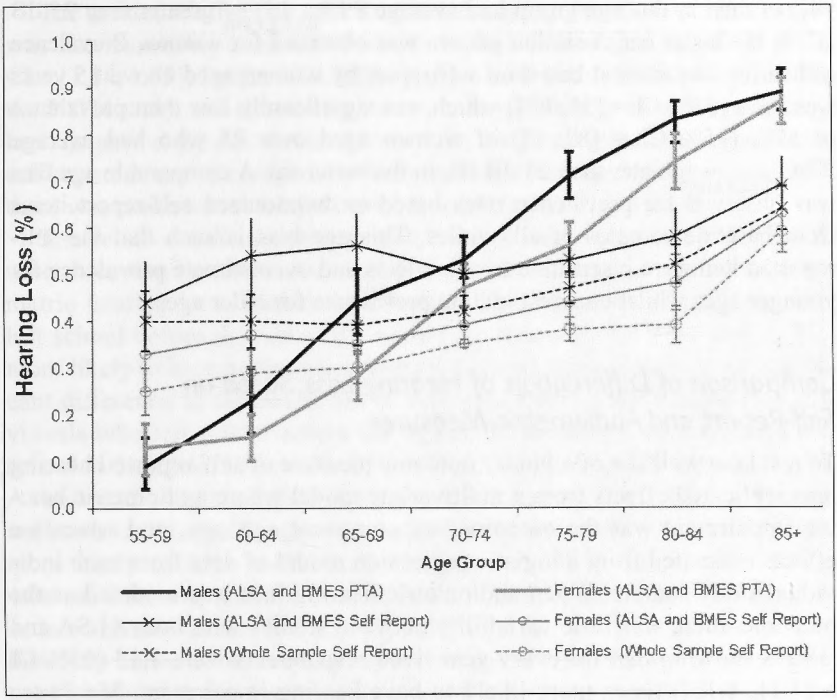

Figure 1. Comparison of prevalence (with 95% CI) of objective ($PTA_{0.5,1,2,4\ kHz}$) and subjective hearing loss by age group and sex at baseline

gradient for measured hearing impairment. The prevalence of self-reported hearing loss increased by only 4.1% for every 5-year increase in age. In contrast, prevalence of measured hearing impairment increased by 13.5% for every 5-year increase in age. Although prevalence rates based on self-reported items were reasonably accurate for adults aged between 65 and 74 years, prevalence based on self-report data greatly overestimated measured hearing loss prevalence for younger age groups but greatly underestimated prevalence rates for older age groups. For example, within the pooled ALSA and BMES sample 44% (95% CI = [35, 53]) of men aged between 55 and 59 reported some degree of hearing loss, whereas only 9% (95% CI = [4, 15]) of men in this age group had average $PTA_{0.5,1,2,4\ kHz}$ greater than 25 dB HL in the better ear. In contrast, 69% (95% CI = [64, 75]) of men aged over 85 years reported some level of hearing difficulty, whereas 89% (95% CI = [85,

94]) of men in this age group had average $PTA_{0.5,1,2,4\ kHz}$ greater than 25 dB HL in the better ear. A similar pattern was observed for women. Prevalence of hearing impairment based on self-report by women aged above 85 years was 63% (95% CI = [56, 69]) which was significantly less than prevalence of 87% (95% CI = [82, 9]) of women aged over 85 who had average $PTA_{0.5,1,2,4\ kHz}$ greater than 25 dB HL in the better ear. A comparable age bias was observed for prevalence rates based on harmonized self-report items from baseline samples of all studies. This age bias is such that the self-reported items are insensitive to age effects and overestimate prevalence for younger ages whilst underestimating prevalence for older ages.

Comparison of Differentials of Hearing Loss Based on Self-Report and Audiometric Measures

To test how well use of a binary outcome measure of self-reported hearing loss replicated effects from a multivariate model where audiometric hearing impairment was the outcome, we compared age, sex, and education effects estimated from a logistic regression model of data from each individual study sample. When audiometric hearing loss was modeled as the outcome, there was little variability between studies with both ALSA and BMES showing that for every year lived respondents were 1.13 (95% CI = [1.11, 1.15]) times more likely to have hearing impairment. Men were 1.47 (95% CI = [1.17, 1.84]) times more likely to have hearing impairment in the ALSA sample, and 1.56 (95% CI = [1.26, 1.91]) times more likely to have hearing impairment in the BMES sample. Although participants who left school before the age of 15 were more likely to have audiometric hearing impairment in both studies, there were differences between these two samples in the magnitude of the OR with early school leavers in the ALSA sample 1.43 (95% CI = [1.14, 1.79]) times more likely to have hearing impairment. Although early school leavers in the BMES sample were 1.17 (95% CI = [0.93, 1.48]) times more likely to have hearing impairment, the OR was not reliably different from 1 ($p = .18$).

When a binary measure of self-reported hearing was modeled as outcome the OR for age was reduced relative to audiometric hearing loss and ranged between 0.98 (95% CI = [0.93, 1.04]) in the PATH study and 1.08 (95% CI = [1.03, 1.12]) in the SOPS study. The OR of 0.98 for the PATH study was in the opposite direction compared to other studies but was not reliably different from 1 ($p = .51$) and could be a result of the narrow-aged cohort for this sample (aged between 64 and 70).

The greater relative risk of self-reported hearing loss associated with men was also reasonably consistent between studies. ORs ranged from 1.51 (95% CI = [1.14, 1.99]) for the MELSHA study, to 1.97 (95% CI = [1.66, 2.34]) for the PATH study, and showed that men are more likely to experience hearing loss. In general the association between sex and hearing loss was stronger for self-report items in comparison with audiometric hearing impairment, and there was consistent overlap of confidence intervals for all studies.

The relative risk of self-reported hearing impairment associated with school-leaving age, was also incongruent between studies and with audiometric hearing loss. Within the baseline ALSA sample, participants who left school before the age of 15 were 1.43 times (95% CI = [1.14, 1.79]) more likely to have audiometric hearing loss, whereas there was no significant difference in the likelihood of reporting hearing difficulties for individuals who left school before the age of 15 compared to those who left school at the age of 15 or older (OR = 0.96, 95% CI = [0.80, 1.15], $p = .69$). A similar, although less divergent pattern was observed in BMES. Although not statistically significant, early school leavers in BMES were 1.17 times more likely to have audiometric hearing loss (95% CI = [0.92, 1.48]), whereas there was no difference in self-reported hearing loss between early or late school leavers (OR = 0.99, 95% CI = [0.79, 1.24]). Of the studies that only collected self-report hearing data, all ORs trended to be greater than 1 indicating that participants who left school before age 15 were more likely to report hearing difficulties, however these effects were statistically significant only for ALSWH (OR = 1.14, 95% CI = [1.05, 1.23]) and MELSHA (OR = 1.41, 95% CI = [1.07, 1.87]).

Discussion

This study aimed to report on the harmonization of dichotomized self-reported hearing loss items and evaluate them against audiometric hearing loss. We compared prevalence estimates derived from self-reported hearing measures with estimates derived from standardized audiometric data in the Australian population. Analyses showed that within the ALSA and BMES studies, a dichotomized measure of self-reported hearing loss appeared to be reasonably sensitive and displayed moderate associations with audiometric assessment. Self-report data, however, did not provide a reliable basis for estimating prevalence in the general population, and depending on the sample characteristics or question wording, had conflicting sensitivity and specificity. In particular, self-reported hearing appeared to overestimate hearing impairment ($PTA_{0.5,1,2,4\,kHz} > 25$ dB HL) in younger age cohorts whereas

underestimating hearing impairment in older age cohorts. Although previous comparisons of self-report with audiometric measures have supported conclusions that self-report data may be sufficient for estimating overall prevalence of hearing loss, these studies only compared prevalence in broad age cohorts and failed to consider an age bias in self-reported health measures (Gates, Cooper, Kannel, & Miller, 1990; Nondahl et al., 1998). Indeed, in this study, the difference between self-report and audiometric-based prevalence for all adults aged 55 years and older was minimal.

Social comparison theory (Willis, 1981) provides one explanation for the failure of self-reported hearing to detect age differences. Social comparison theory maintains that older adults tend to overrate their perceived health because they make implicit downward comparisons with negative old-age stereotypes (Heckhausen & Brim, 1997; Sargent-Cox, Anstey, & Luszcz, 2008). The age bias inherent in self-reported hearing items could therefore reflect the downward social comparisons older adults are surmised to make when rating their health despite loss of functioning (Heckhausen & Brim, 1997). A similar explanation was given for the poorer performance of the HHIE in estimating prevalence in adults aged 65 years and older compared to adults aged between 48 and 64. Nondahl et al. (1998) suggested that older adults are more likely to be accepting of hearing impairment as they do not consider it an unusual aspect of ageing. The high prevalence and common experience leads to hearing loss becoming normalized in older adults. Furthermore, as hearing decline is generally a gradual process, many adults have time to adjust to hearing loss.

Younger age groups could overestimate their hearing difficulties for a number of reasons. First, they are more likely to be actively participating in the workforce and have greater work-related demands on their hearing. Certainly after the retirement age of 65, self-report data no longer overestimates audiometric hearing impairment. Also, hearing ability for pure-tone thresholds below 4kHz begin to decline in the 50s, and initial losses may be more noticeable at these ages (Wiley, Chappell, Carmichael, Nondahl, & Cruickshanks, 2008). Low levels of hearing aid utilization in younger age groups could also contribute to the differences between self-report and audiometric measures. It has been reported that adults may experience hearing difficulties for up to 10 years before they recognize their hearing to be a problem and access hearing services during their mid 70s (Davis, Smith, Ferguson, Stephens, & Gianopoulos, 2007). Finally, this could reflect a cohort effect whereby younger cohorts are more likely to report health problems and functional difficulties. It should not be discounted that the apparent overreporting to hearing loss is a real effect reflecting the poorer health status of younger

cohorts. Seeman, Merkin, Crimmins and Karlamangla (2010) found significant increases in disability over a 16-year period in a cohort aged 60-69 years whereas those aged 70-79 years showed no significant changes in disability and those aged 80 years and above showed lower prevalence of functional limitations.

Hearing impairment was more likely to occur in men, older adults, and early school leavers regardless of whether hearing impairment was defined by pure-tone thresholds or self-report. However, when estimates were based on self-report items rather than audiometric measures, there were considerable differences in the strength of these associations and there was more variability between studies. In particular, self-reported hearing items were not sensitive to the relation between age left school and hearing impairment. Self-reported hearing items consistently underestimated the strength of the age-related risk of hearing impairment and tended to overestimate the association between sex and hearing impairment. It is possible that some of the discrepancy in findings related to the self-reported hearing impairment items was due to design difference in the contributing studies, including sample composition and age structure.

Of particular interest is the influence of the response scale on the reliability of self-reported hearing loss. Two almost identical questions: "do you have any difficulty with your hearing?" (Binary response: *yes*, *no*) and "do you have difficulties with your hearing" (three level: *yes*, *sometimes*, *no*) were shown to have conflicting associations with measured hearing loss. The latter item is of a similar form to the HHIE (Ventry & Weinstein, 1982), and was used as a screening question prior to implementing the HHIE. Binary responses and responses framed in reference to the grade of hearing difficulty (none, slight, moderate severe) were good predictors of measured hearing loss. In contrast, nonbinary responses framed in reference to the frequency or duration of hearing difficulty (*yes*, *sometimes*, *no*) was extremely poor predictors of measured hearing loss and not at all associated with audiometric assessment. This suggests such measures are not an adequate alternative to audiometric screening and that the context in which a question is asked may also influence response propensities.

Detailed, self-reported hearing loss could be useful in screening for hearing impairment, and in accounting for the multiple, independent, impacts now described for this sensory impairment (Gopinath et al., 2010; Gopinath, 2009b; Karpa et al., 2010; Mitchell et al., 2009; Schneider et al., 2010; Tay et al., 2006). Hickson et al. (1999) argue that objective and subjective measures may be tapping different disability profiles and that both are needed to select clients for rehabilitation. In their study, 15% of participants classified

with normal hearing by audiometry reported hearing difficulty, whereas 17.5% of those with moderate or greater audiometric hearing loss reported no hearing difficulty. Our findings do not discount the efficacy of self-reported hearing in identifying disability due to hearing loss (Newman, Weinstein, Jacobson, & Hug, 1990). Perceived health is often reported to be a stronger predictor of quality of life and well-being than actual illness. Therefore, self-report items do have clinical utility and play a role in determining social burden of hearing loss.

There were study differences in self-reported hearing loss. Some items made specific reference to hearing ability with a hearing aid, whereas others did not explicitly distinguish between perceived hearing ability either with or without a hearing aid. There were qualitative differences in the nature of hearing loss ("problems," "difficulty," "loss," "adequacy"), temporal reference frame ("ever," "12 months," "currently") and response options ("binary" vs. rating scale of either the "frequency" of hearing problems or "severity" of hearing problems). This variation in self-report measures across studies is indicative of two different measures of hearing loss. Questions referring to "hearing loss" with a binary response are more likely to be perceived as directly asking about physical hearing loss; whereas questions referring to the frequency of hearing difficulties, or require a graded judgment concerning hearing-related problems, may lead the respondent to consider in what contexts and how regularly they experience difficulties due to hearing loss. A comparison of the base-line self-report item with subsequent waves within ALSA also revealed that, despite the sample becoming more homogonous with respect to age, reference to hearing aids reduced the association between self-report and audiometric data. Thus, consideration of hearing difficulties when using a hearing aid (if owned) is also more likely to elicit a response that reflects hearing disability.

The need to dichotomize responses to self-reported hearing loss items when creating harmonized measures resulted in information loss and reduced variability in the data. However, harmonization improves the comparability of data across studies, enabling future investigations into hearing disability using the pooled data set. Pooled analyses of the harmonized self-report variable will require careful interpretation and consideration of how study differences may influence findings.

In summary, our findings suggest that self-reported hearing measures are not sufficiently sensitive to be used to estimate prevalence or incidence of hearing impairment, particularly for adults of working age (below 65 years) and adults aged above 75 years. Instead, these measures may be useful as broad indicators of perceived hearing disability and when investigating

impacts of hearing loss on health and well-being. Given the increase in prevalence of hearing impairment due to population aging, there is a need for epidemiological studies to combine both objective measures of hearing loss with measures that evaluate the impact of hearing loss on functioning and healthy aging.

Acknowledgments

The authors would like to thank Julie Byles, Mary A. Luszcz, and Olivier Piguet. They would like to thank the participants for volunteering their time to be involved in the respective studies. Details of all studies contributing data to DYNOPTA, including individual study leaders and funding sources, are available on the DYNOPTA website (http://DYNOPTA.anu.edu.au).

Authors' Note

The data on which this research is based were drawn from several Australian longitudinal studies including the following: the Australian Longitudinal Study of Ageing (ALSA), the Australian Longitudinal Study of Women's Health (ALSWH), the Blue Mountain Eye Study (BMES), the Canberra Longitudinal Study of Ageing (CLS), the Melbourne Longitudinal Studies on Healthy Ageing (MELSHA), the Personality And Total Health Through Life Study (PATH), and the Sydney Older Persons Study (SOPS). These studies were pooled and harmonized for the Dynamic Analyses to Optimise Ageing (DYNOPTA) project. The findings and views reported in this article are those of the author(s) and not those of the original studies or their respective funding agencies.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This work was supported by the National Health and Medical Research Council (NHMRC) Grant # 410215. Professor Anstey is funded by NHMRC Fellowship #366756.

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Journal of Gerontology: MEDICAL SCIENCES
Cite journal as: J Gerontol A Biol Sci Med Sci
doi:10.1093/gerona/gls066

Cognitive, Health, and Sociodemographic Predictors of Longitudinal Decline in Hearing Acuity Among Older Adults

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Background. We aimed to investigate predictors of change in pure-tone hearing thresholds in older adults.

Methods. Data were drawn from a pooled sample from the Dynamic Analyses to Optimise Ageing (DYNOPTA) project ($N = 4,221$, mean age = 73.6, range: 50–103 years). Pure-tone hearing thresholds were tested for frequencies between 0.5 and 8 kHz, on up to four occasions over a period of 11 years. Linear mixed models tested for predictors of change in hearing.

Results. Hearing loss for high-range frequencies preceded decline in low-range frequencies. Men had higher baseline hearing thresholds, but women experienced faster rates of decline in hearing for mid- to high-range frequencies. The estimated rate of change for a 75-year-old adult was 0.91 decibel hearing level (dB HL) per year for pure-tone thresholds averaged over frequencies ranging between 0.5 and 4 kHz in the better ear. Baseline age ($\beta = 0.03$, $p < .01$), hypertension ($\beta = 0.15$, $p < .01$), and probable cognitive impairment ($\beta = 0.40$, $p = .01$) were independent predictors of annual rate of change in hearing thresholds. Incidence of probable cognitive impairment was also associated with higher hearing thresholds. Other known correlates for prevalence of hearing impairment, including low education, noise damage, diabetes, and history of stroke were independently associated with baseline levels of hearing but were not predictive of change in hearing thresholds.

Conclusions. Faster rates of decline in hearing are predicted by probable cognitive impairment and hypertension.

Key Words: Presbycusis—Age-related hearing loss—Cognitive impairment—The Australian Longitudinal Study of Ageing—The Blue Mountains Eye Study.

Received April 18, 2011; Accepted January 24, 2012

Decision Editor: Luigi Ferrucci, MD, PhD

AGE-RELATED hearing loss is highly prevalent among older adults (1–4). It features among the leading causes of years lived with disability and is considered a substantial contributor to global burden of disease (5). Cross-sectional studies have identified diabetes (6,7), cardiovascular disease, hypertension, and blood pressure (8) as risk factors for hearing loss. Hearing loss has also been linked with poor physical and mental health, falls (9), mortality (10,11), and lower cognitive functioning or dementia (12–19). However, longitudinal analyses have failed to show an association between many of these risk factors with incidence of age-related hearing loss (20–22).

Divergent patterns of predictors for prevalence versus rates of decline in hearing have been suggested to arise from methodological factors. These include insufficient statistical power, differences in the rate of onset, and age dependency of hearing loss (20). Alternatively, the common practice of dividing ranges of averaged hearing thresholds into conventional categories of hearing loss (eg, no impairment, mild impairment, moderate impairment) may obscure true associations between risk factors for change in hearing acuity. We address these issues by employing growth curve techniques to examine hearing trajectories in a larger representative sample of older adults than has previously been available. Other studies investigating longitudinal changes in continuous measures have primarily focused on mapping age and sex trajectories of individual pure-tone frequencies (23–27). This study aims to extend the current understanding of age-related hearing loss by additionally investigating sociodemographic and health-related risk factors for change in hearing thresholds.

Methods

Participants

Data were drawn from the Australian Longitudinal Study of Ageing (ALSA) (28) and the Blue Mountains Eye Study

(BMES) (12) as part of the Dynamic Analyses to Optimise Ageing (DYNOPTA) project (29). ALSA drew a random sample of adults aged 70 years and older from the electoral role for the Adelaide metropolitan area of South Australia. ALSA oversampled men aged 85 years and older and also recruited spouses aged 65 years and older, or others over 70 years who were cohabiting with the sampled participant. Data collection pertinent to the aims of this study occurred within ALSA at wave 1 (1992), wave 3 (1994), wave 6 (2000–2001), and wave 7 (2003–2004). BMES attempted to recruit all adults aged 49 years and older from two postcodes in Blue Mountains region west of Sydney, Australia. Data collection pertinent to the aims of this study occurred within BMES occurred at wave 2 (1997–1999) and wave 3 (2002–2004). We define the baseline sample as pooled data from wave 1 of ALSA and wave 2 of BMES.

Measures

Audiometric testing was conducted by a trained interviewer in each study. Hearing loss was assessed by uncorrected pure-tone thresholds in each ear at frequencies of 0.5, 1, 2, 3, 4, 6, and 8 kHz using calibrated portable audiometers for ALSA participants and in a sound-treated booth for BMES participants. Outcome variables used in analyses reported in this study were pure-tone thresholds in the better ear and a pure-tone average (PTA) of low- to mid-range frequencies important for speech perception (0.5, 1, 2, and 4 kHz) in the better ear. Thresholds ranged between 0 and 120 decibel hearing level (dB HL), thresholds of 120 dB HL were treated as outliers and coded as missing values. In BMES, frequencies of 3 kHz were only tested in participants with a difference of 20 dB HL between the 2 and 4 kHz frequencies.

Medical conditions were obtained by self-report of clinician diagnoses and included: diabetes, hypertension, history of stroke, and history of heart attack. Corrected visual acuity was tested with a logMAR chart at a distance of 3 m, with visual impairment defined by values greater than 0.3 logMAR. A score of 23 or less on the Mini-Mental State Examination (30) was used as an indicator of probable cognitive impairment. Smoking status was also obtained by self-report.

Information on workplace noise exposure was collected in ALSA with the question "Have you ever worked in a noisy environment where you had to shout to be heard?" and in BMES with the question "Have you ever worked in a noisy industry or noisy farm environment?" To identify cases with likely noise induced hearing loss, high-frequency audiometric noise notches were defined using the criteria described by Coles and colleagues (31). These criteria have been shown to have strong agreement with expert consensus (32).

Analysis

For descriptive purposes, the mean and standard deviation $PTA_{0.5,\,1,\,2,\,4\ kHz}$ were calculated for 10-year age groups and for each covariate. Linear mixed models were used to estimate trajectories of hearing thresholds in the better ear. All analyses included random effect variance components for the intercept and slope (time) with an unstructured covariance matrix. The optimal scaling of time was ascertained by comparing Bayesian Information Criterion (BIC) values for models that indexed time as linear and quadratic functions of age, with models that indexed time over a "years in study" metric adjusting for age at baseline with an interaction term between age at baseline and years in study. Better model fit is indicated by lower BIC values. Age and sex trajectories of hearing thresholds in the better ear were then estimated for each tone frequency and $PTA_{0.5,\,1,\,2,\,4\ kHz}$. Model coefficients were used to graph the mean trajectories for men and women aged 60, 75, and 90 years at baseline. The predicted mean ages at which the $PTA_{0.5,\,1,\,2,\,4\ kHz}$ trajectory crossed thresholds of 25 and 40 dB HL were estimated for men and women by solving the model equation for "time."

Interaction terms between baseline predictors and time tested between-person differences in hearing trajectories. We included baseline predictors of age (mean centered to 75 years), sex (female = 1), and indicators of probable cognitive impairment, diabetes, stroke, hypertension, visual impairment, and smoking status. Time invariant predictors were workplace noise exposure, high-frequency audiometric noise notches, and sociodemographics. For those baseline conditions that were significantly associated with change in hearing thresholds, we also included an indicator of post-baseline incidence to test if incident medical conditions were also associated with hearing loss. A four-stage procedure was employed to evaluate predictors of change in $PTA_{0.5,\,1,\,2,\,4\ kHz}$. In the first stage, we conducted a series of univariate models that estimated unadjusted associations between each predictor variable with baseline hearing levels and longitudinal hearing trajectories. In the second step, we ran the same set of univariate models adjusting for age at baseline. We then estimated a full multivariate model that included all covariates. In the final step, BIC were used to evaluate the multivariate model, which was refined by excluding model terms that did not contribute to the overall model fit. In order to determine the extent to which noise damage confounded inferences concerning age-related hearing loss, multivariate analyses were repeated excluding all participants who reported 5 years of workplace-related noise exposure or were identified to have high-frequency noise notches. All analyses were conducted using Stata version 10 (33).

Results

Description of Sample Characteristics

The baseline sample profile is described in Table 1. The pooled sample comprised 4,221 participants (46.3% men) with a mean age of 73.6 years (SD = 8.9, range = 50–103). A total of 366 participants were classified with probable cognitive impairment at baseline, with a further 274 incident

Table 1. Baseline Sample Profile, 3,526 Australian Adults Aged 50 and Older

	N	%	PTA (dB) Mean (SD)
Sex			
Men	1,633	46.3	30.6 (15.7)
Women	1,893	53.7	26.0 (14.7)
Age (y)			
50–59	285	8.1	15.2 (11.3)
60–69	861	24.4	20.8 (13.4)
70–79	1,562	44.3	28.7 (13.1)
80–89	750	21.3	38.5 (14.2)
90+	68	1.9	46.6 (17.0)
Hearing loss			
Normal	1,718	48.7	16.0 (5.9)
Mild	1,140	32.3	32.4 (4.2)
Moderate–severe	668	18.9	52.0 (11.8)
Qualification			
Secondary only	1,647	46.7	29.7 (15.2)
Postsecondary	1,442	40.9	26.5 (15.1)
Tertiary	242	6.9	25.9 (14.2)
Occupation			
Tradesperson	440	12.5	32.9 (17.1)
Plant, machine operators, and drivers	129	3.7	31.6 (16.1)
Laborers and related workers	231	6.6	31.7 (16.1)
Other	2,726	77.3	26.9 (14.7)
Smoking status			
Never	1,741	49.4	27.7 (15.7)
Former	1,458	41.3	28.8 (14.8)
Current	291	8.3	26.9 (15.7)
Workplace noise exposure			
<1 y	2,339	66.3	27.1 (14.8)
1–5 y	323	9.2	29.5 (16.0)
5+ y	864	24.5	30.6 (16.1)
Hearing aid			
Yes	401	11.4	49.7 (15.1)
Hearing restricts social life			
Never	2,143	60.8	25.2 (13.9)
Sometimes	431	12.2	38.3 (14.4)
Often	209	5.9	47.3 (20.1)
Medical conditions (self-report)			
Diabetes	252	7.1	31.4 (16.6)
Stroke	151	4.3	32.4 (16.3)
Heart attack	353	10.0	31.3 (15.3)
Hypertension	1,234	35.0	27.4 (14.7)
Any circulatory condition	1,729	49.0	29.0 (15.3)
Measured conditions			
Systolic > 145 mmHg	2,334	66.2	28.3 (15.2)
Diastolic > 95 mmHg	428	12.1	26.1 (15.2)
Visual acuity > 0.3 logMAR	507	14.4	35.0 (15.9)
MMSE < 24	218	6.2	38.9 (16.7)

Note: logMAR = logarithm of the minimum angle of resolution; MMSE = Mini-Mental State Examination; PTA = Pure-tone average (dB) of 0.5, 1, 2, 4 kHz in the better ear. Column percentages are based on the number of participants who gave a valid response, rows may not sum to whole sample due to missing data.

cases in subsequent waves. There were 211 participants identified with high-frequency audiometric noise notches at any time (mean baseline age = 69.9, 75.4% men), and 851 participants reported workplace-related noise exposure for 5 or more years.

The average time intervals between successive waves were 3.8 (SD = 1.8), 6.1 (SD = 0.2), and 3.1 (SD = 0.2) years, with participants providing an average of 2 waves of data. Prior to the commencement of wave 2, 16.6% of participants were lost to attrition and a further 6.4% were deceased. The BMES sample (n = 2,334) only provided data for waves 1 and 2. Within the ALSA sample, 44.5% of baseline participants were deceased at wave 3, this increased to 58.8% at wave 4.

Audiometric testing was completed by 3,526 participants at baseline ($PTA_{0.5, 1, 2, 4\ kHz}$ Mean (M) =28.2 dB, SD = 15.2) and 3,011 participants at wave 2 (M = 30.1 dB, SD = 15.5). Based on the ALSA sample, $PTA_{0.5, 1, 2, 4\ kHz}$ data were available for 525 participants at wave 3 (M = 37.0 dB, SD = 14.3) and 391 participants at wave 4 (M = 38.6 dB, SD = 15.3).

Modeling of Time

Linear mixed models that indexed time over a years in study metric and adjusted for baseline age (BIC = 54,272.0) provided a better description of longitudinal change in $PTA_{0.5, 1, 2, 4\ kHz}$ and were preferable to models that indexed time using an "age" metric (BIC = 55,195.9). This was consistent with previous recommendations regarding the optimal scaling of time in longitudinal analyses with broad age cohorts (34). All subsequent results index time over a years in study metric.

Trajectories of Hearing Thresholds for Men and Women

The estimated age-related trajectories for each of the seven pure-tone frequencies and $PTA_{0.5, 1, 2, 4\ kHz}$ in men and women are presented in Figure 1. An increase in hearing thresholds over time indicates a decline in hearing acuity. Relative to high-range frequencies, change in hearing thresholds for low-range frequencies began later and accelerated with age. Age-related changes in frequencies greater than 4 kHz were observed for adults of all ages, whereas frequencies of 0.5 and 1 kHz did not show marked increases in pure-tone thresholds until individuals were aged in their 70s. There were no sex differences in rate of change in hearing for $PTA_{0.5, 1, 2, 4\ kHz}$ and low-range frequencies. However, women had lower intercepts and faster increases in thresholds greater than 3 kHz. Sex differences in intercepts and slopes were greatest for mid-range frequencies. For adults aged 75 years at baseline, the estimated mean $PTA_{0.5, 1, 2, 4\ kHz}$ trajectory crossed a threshold of 25 DB HL (often defined as mild hearing impairment) at ages 67.8 years for men and 71.1 years for women. The estimated mean $PTA_{0.5, 1, 2, 4\ kHz}$ trajectory crossed a threshold of 40 dB HL (often defined as moderate hearing impairment) at ages 83.2 years for men and 86.5 years for women.

Predictors of Hearing Loss

Table 2 shows the results from the series of univariate-, age-, and multivariate-adjusted linear mixed models for $PTA_{0.5, 1, 2, 4\ kHz}$ in the better ear. In the age-adjusted univariate models, all baseline covariates reliably predicted initial

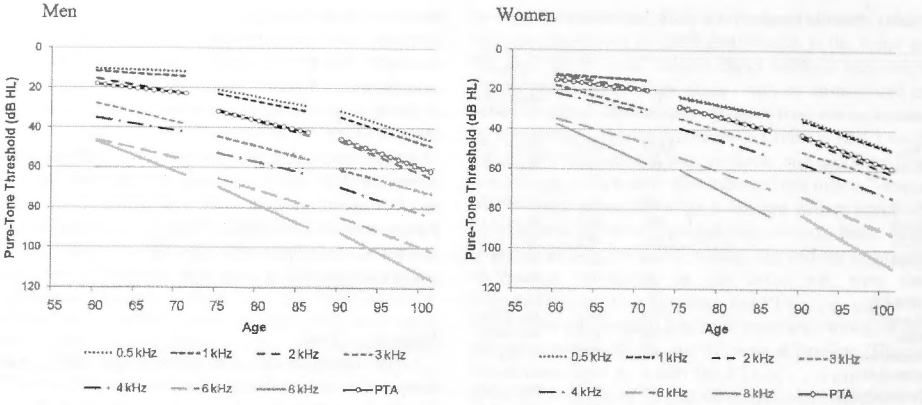

Figure 1. Unadjusted 11-year trajectories of pure-tone thresholds decibel hearing level (dB HL) for frequencies of 0.5, 1, 2, 3, 4, 6, and 8 kHz in the better ear, and $PTA_{0.5,\,1,\,2,\,4\ kHz}$ in the better ear, estimated for three cohorts of men (left panel) and women (right panel) aged 60, 75, and 90 years at baseline. The y-axis has been reversed so a negative gradient indicates a decline in hearing performance. Sample excludes participants with high-frequency noise notches. The better ear was defined by $PTA_{0.5,\,1,\,2,\,4\ kHz}$.

levels of $PTA_{0.5,\,1,\,2,\,4\ kHz}$. However, the only statistically significant predictors of rate of change were baseline age, sex, workplace noise exposure, and probable cognitive impairment. Faster increases in hearing thresholds were observed for older adults, women, and participants with probable cognitive impairment. Interestingly, noise notches were not associated with hearing trajectories, but participants reporting 5 years or more of workplace noise exposure showed slower increases in hearing thresholds.

In multivariate analyses, smoking, visual impairment, and postsecondary nontertiary qualifications did not contribute to overall model fit and were excluded from the final model. For an adult aged 75 years, the average $PTA_{0.5,\,1,\,2,\,4\ kHz}$ trajectory increased at a rate of 0.86 dB HL per annum, with annual increase in the rate of change of 0.03 dB HL. After adjusting for sociodemographic and health variables, there were no sex differences in rate of change in hearing, though probable cognitive impairment at baseline was associated with both poorer initial $PTA_{0.5,\,1,\,2,\,4\ kHz}$ levels ($\beta_{level} = 3.91$, 95% confidence interval [CI] = 2.05–5.77) and faster rates of change in $PTA_{0.5,\,1,\,2,\,4\ kHz}$ ($\beta_{change} = 0.40$, 95% CI = 0.12–0.68). Incident probable cognitive impairment was also associated higher $PTA_{0.5,\,1,\,2,\,4\ kHz}$ ($\beta_{incident} = 0.83$, 95% CI = 0.12–1.55). Probable cognitive impairment at baseline was not associated with change in better ear thresholds for individual frequencies greater than 4 kHz. Multivariate analyses also revealed greater rates of change in thresholds for participants reporting clinically diagnosed hypertension at baseline ($\beta_{change} = 0.15$, 95% CI = 0.06–0.25). Excluding participants who reported 5 years or more of workplace noise exposure or who had high-frequency noise notches, resulted in only minor adjustments to model coefficients and the substantive findings remained unchanged (data not shown).

Discussion

This study reports on patterns and predictors of change in 11-year trajectories for hearing thresholds in older adults. Hearing loss for frequencies important for speech perception increased at an average rate of 0.91 dB/year. Unsurprisingly, these rates of hearing decline were accelerated for older ages. Half of all adults in the oldest old cohort, aged 85 years and older, had moderate hearing loss, and almost all of the oldest old cohort could be expected to have at least a mild degree of hearing loss. A key finding is that cognitive impairment was independently associated with lower levels and accelerated declines in peripheral hearing ability. Furthermore, incidence of cognitive impairment was also associated with poorer hearing. Thus, both between-person differences and within-person change in cognitive function were identified as risk factors for hearing loss. Hypertension was also found to be predictive of greater decline rates in hearing.

This study adds to the growing literature linking poor hearing with neurocognitive disorders (13–18) and age-related cognitive decline (19). Early hearing loss and rapid hearing decline have been suggested to be precursors of dementia and could be useful risk markers in dementia diagnosis (16,18), though the analyses presented here do not test this hypothesis. Rather than assessing hearing loss as a leading indicator of cognitive decline, we show that individuals with cognitive impairment experience faster declines in peripheral hearing. That cognitive impairment was not predictive of decline in high-frequency thresholds suggests underlying mechanistic pathways. However, the mechanism for this is unclear and cannot be identified from this study. The co-occurrence of cognitive impairment and hearing loss should be expected due to their associations with aging, but further explanation is warranted because their association

Table 2. Fixed Effects for Predictors of Baseline Levels and Longitudinal Trajectories of Hearing Thresholds ($PTA_{0.5, 1, 2, 4\ kHz}$) in the Better Ear Estimated From Univariate and Multivariate Linear Mixed Models

	Univariate Models		Age-Adjusted Models		Multivariate (full model)		Multivariate (final model)	
	β (SE)	*p*	β (SE)	*p*	β (SE)	*p*	β (SE)	*p*
Unadjusted								
Intercept (baseline)	27.91 (0.26)	<.01	29.92 (0.23)	<.01	27.82 (0.96)	<.01	28.69 (0.48)	<.01
Time (y)	0.84 (0.03)	<.01	0.97 (0.03)	<.01	0.89 (0.10)	<.01	0.86 (0.03)	<.01
Demographics								
$Age_{baseline}$*	0.91 (0.03)	<.01	0.91 (0.03)	<.01	0.87 (0.03)	<.01	0.89 (0.03)	<.01
$Age_{baseline}$ by time	0.03 (0.00)	<.01	0.03 (0.00)	<.01	0.03 (0.00)	<.01	0.03 (0.00)	<.01
Women	−4.64 (0.51)	<.01	−3.32 (0.44)	<.01	−2.04 (0.54)	<.01	−1.54 (0.48)	<.01
Women by time	0.09 (0.05)	0.07	0.12 (0.05)	.02	0.10 (0.05)	0.08	Dropped from model	
Cognitive Status								
$MMSE < 24_{baseline}$	11.75 (1.04)	<.01	5.16 (0.92)	<.01	3.34 (1.02)	<.01	3.91 (0.95)	<.01
$MMSE < 24_{baseline}$ by time	0.54 (0.15)	<.01	0.37 (0.14)	.01	0.47 (0.15)	<.01	0.40 (0.14)	.01
$MMSE < 24_{incidence}$	1.55 (0.36)	<.01	0.93 (0.36)	.01	0.87 (0.39)	.03	0.83 (0.36)	.02
Qualifications								
Secondary only	3.91 (1.04)	<.01	2.14 (0.90)	.02	2.37 (0.93)	.01	1.08 (0.45)	.02
Secondary only by time	0.05 (0.10)	.64	−0.05 (0.10)	.61	−0.12 (0.09)	.20	Dropped from model	
Postsecondary	0.87 (1.05)	.41	1.35 (0.90)	.14	1.23 (0.92)	.18	Dropped from model	
Postsecondary by time	−0.02 (0.10)	.83	−0.04 (0.10)	.68	−0.08 (0.09)	.42	Dropped from model	
Smoking Status								
Former smoker	1.19 (0.54)	.03	0.89 (0.46)	.05	−0.45 (0.51)	.38	Dropped from model	
Former smoker by time	−0.04 (0.05)	.41	−0.05 (0.05)	.31	0.03 (0.05)	.62	Dropped from model	
Current smoker	−1.03 (0.97)	.29	2.07 (0.83)	.01	0.24 (0.88)	.79	Dropped from model	
Current smoker by time	−0.14 (0.10)	.17	−0.03 (0.10)	.79	0.11 (0.10)	.24	Dropped from model	
Workplace noise exposure								
5 y or more	3.51 (0.61)	<.01	4.96 (0.51)	<.01	3.80 (0.59)	<.01	3.97 (0.57)	<.01
5 y or more by time	−0.23 (0.06)	<.01	−0.18 (0.06)	<.01	−0.07 (0.06)	.27	−0.13 (0.05)	.01
1–5 y	2.49 (0.90)	.01	3.88 (0.76)	<.01	3.48 (0.83)	<.01	3.27 (0.78)	<.01
1–5 y by time	−0.01 (0.09)	.87	0.01 (0.09)	.90	<.01 (0.08)	.97	Dropped from model	
Noise notch								
Notch	1.29 (0.59)	.03	1.61 (0.59)	.01	0.78 (0.57)	.17	1.24 (0.49)	.01
Notch by time	−0.01 (0.18)	.97	−0.01 (0.17)	.94	−0.04 (0.17)	.80	Dropped from model	
Medical conditions								
Hypertension	−1.38 (0.54)	.01	−0.93 (0.46)	.04	−0.77 (0.49)	.11	−0.79 (0.47)	.09
Hypertension by time	0.11 (0.05)	.04	0.10 (0.05)	.06	0.14 (0.05)	<.01	0.15 (0.05)	<.01
Diabetes	3.14 (1.01)	<.01	3.06 (0.86)	<.01	2.76 (1.14)	.02	2.09 (0.85)	.01
Diabetes by time	−0.09 (0.11)	.43	−0.06 (0.11)	.54	−0.23 (0.14)	.11	Dropped from model	
Stroke	4.67 (1.29)	<.01	3.28 (1.10)	<.01	2.66 (0.90)	<.01	2.56 (1.10)	.02
Stroke by time	−0.19 (0.16)	.22	−0.16 (0.15)	.29	−0.06 (0.10)	.56	Dropped from model	
Visual impairment	8.66 (0.72)	<.01	2.04 (0.66)	<.01	1.31 (0.67)	.05	Dropped from model	
Visual impairment by time	0.13 (0.08)	.08	−0.04 (0.08)	.59	−0.10 (0.07)	.15	Dropped from model	

Notes: $MMSE < 24_{baseline}$ = baseline probable cognitive impairment; $MMSE < 24_{incidence}$ = incidence of probable cognitive impairment post-baseline. Random effects for intercept and slope are not shown. Reference group for each variable: Men: No cognitive impairment, tertiary qualified, never smoker, less than 1 year noise exposure, absent noise notch, no reported hypertension, no reported diabetes, no reported stroke, and no visual impairment.

*$Age_{baseline}$ is centered to 75 years.

remains after statistically controlling for the effects of age. A third variable not properly adjusted for in this study, such as cerebral microangiopathy, is the most likely explanation for the association between cognition and hearing decline. As dementia pathology is not believed to affect the inner ear or cochlea (35), the current findings could simply be accounted for by top-down processing effects and reflect a more cautious or impaired decision-making process regarding tone perception judgments. Older adults, particularly those with poor executive functioning, may show a response bias whereby greater certainty is required before they acknowledge an audible tone. To a lesser extent, these findings could partially be explained by difficulties experienced by people with sensory loss when completing standard neuropsychological assessments. However, such explanations can generally be discounted as it is possible to conduct audiometric testing in young children, and trained clinical interviewers should be sensitive to hearing limitations of study participants (16).

A combination of histological, electrophysical, and molecular mechanisms in both the peripheral and central nervous system underlie hearing loss (36). It is likely that any biological mechanism underlying a link between dementia and hearing loss occurs centrally upstream of the cochlea (18). For example, Alzheimer's disease pathology has been observed in auditory system pathways such as the ventral

nucleus of the medial geniculate body and in the auditory cortex, but not in cochlear nuclei (35). As unaided pure-tone thresholds were used in this study, we are unable to draw direct inferences about the association between cognitive function and central auditory processing. Our understanding of the temporal interrelations between hearing and cognition would be improved by longitudinal analyses of specific cognitive domains, hearing thresholds, and hearing measures that better asses central presbycusis and neural loss, such as dichotic listening or synthetic sentence identification tasks (2).

Our results support previous findings where risk factors for prevalence of hearing loss, including smoking, diabetes, and stroke (20–22), were not found to be predictive of incidence of hearing loss. Even cross-sectional associations between these factors and hearing loss remain in question. Recent analyses of 717 older adults in the National Health and Nutritional Examination Survey (4) failed to find independent associations between low-frequency, speech-frequency, or high-frequency thresholds with the same set of risk factors, regardless of whether thresholds were modeled as continuous or binary outcomes. This contrasts with our findings, as both diabetes and stroke were cross-sectionally associated with poor baseline hearing. These inconsistencies could arise from methodological differences and the larger sample available in DYNOPTA. Lin and colleagues (4) also speculate that smoking, diabetes, and other cardiovascular risk factors may only have weak associations with hearing loss that are mediated or obscured by other factors. It is therefore intriguing to note the opposite pattern of results for hypertension, which was not predictive of baseline hearing levels but was a risk factor for change. The relation between hypertension and hearing loss is uncertain. Although some researchers have identified hypertension as being linked with hearing loss (2), in particular systolic blood pressure (37), this was not the case in the National Health and Nutritional Examination Survey (4). This deserves further investigation.

Age-related declines in sensory functioning have multiple etiologies, ranging from genetic factors (38) to environmental exposures (36,39), but it has been argued recently that between-person differences in audiometric hearing thresholds can be primarily attributed to genetic variation (40). If so, then this may explain why there has been a failure to show an association between changes in hearing performance with many of the known risk factors for poor hearing. The inability to identify predictors for change in hearing and the equivocal cross-sectional findings suggest that rate of hearing decline may be a better indicator of putative normative or primary ageing processes and less influenced hy disease than other functions. If higher intercepts reflect earlier onset of decline, this could indicate that hearing loss may begin at earlier ages for individuals with poor health, but the rate of hearing loss remains stable for most groups, with the exception of individuals with cognitive impairment or hypertension.

Paradoxically, there was no evidence of a relation between audiograms indicative of noise damage with hearing trajectories, yet noise exposure was predictive of more gradual declines in hearing. This is not completely inconsistent with a previous study that demonstrated slower hearing change for frequencies between 3 and 6 kHz, yet accelerated change for adjacent frequencies of 2 and 8 kHz, among individuals with noise notches (41). These findings were based on a younger sample of men and a different methodology to that employed in the current study. Our failure to identify high-frequency noise notches as a risk factor for change could be due to the difficulty in reliably identifying notches in older adults, particularly for ages when noise-induced hearing loss becomes concomitant with age-related hearing loss (31).

Our results are consistent with existing knowledge about the general progression of age-related hearing loss (2). Typically, age-related hearing loss begins with loss of the ability to perceive high frequencies, then gradually extends to low-range frequencies. High-frequency hearing loss has previously been reported to begin during the 50s (23), so it is likely that decline for high frequencies began before study commencement. Although men had poorer hearing levels for mid- and high-range frequencies, women experienced faster rates of hearing decline for these ranges. The lower initial levels for men probably reflect an earlier age onset of hearing loss.

Differential patterns of hearing loss occur across a spectrum of tone frequencies, which can be either independent of or related to age (42). Due to the time intervals between hearing measurements, we lacked the data to detect rapid declines that occurred independently of age effects over a short time frame. At least four distinct types of presbycusis have been classified, each characterized by a unique pattern of change (36,43), which we were also unable to investigate here. This study has not included ototoxic agents (3,36), and genetic data were not available. We also lacked clinical diagnoses of dementia. These caveats notwithstanding, ours is the largest data set to assess the predictors of hearing loss.

In summary, this study contributes to existing knowledge of the association between impaired cognitive function and hypertension with accelerated decline in hearing. Our findings highlight the need for researchers and clinicians to be aware of impaired cognitive functioning when assessing hearing performance, and conversely, of hearing limitations when diagnosing, screening, and managing individuals with dementia or other cognitive impairments. With the projected rise in the age-adjusted prevalence of hearing loss, its relation to health, well-being, and longevity, there is a need for greater awareness and a better understanding of the development of age-related hearing loss and its interaction with comorbid chronic health conditions.

FUNDING

This work was supported by the National Health and Medical Research Council (NHMRC) grant no. 410215. Professor K.J.A. is funded by NHMRC Fellowship no. 1002560.

ACKNOWLEDGMENTS

The data on which this research is based were drawn from the ALSA and the BMES. These studies were pooled and harmonized for the Dynamic Analyses to Optimise Ageing (DYNOPTA) project. All studies would like to thank the participants for volunteering their time to be involved in the respective studies. Details of all studies contributing data to DYNOPTA, including individual study leaders and funding sources, are available on the DYNOPTA website (http://DYNOPTA.anu.edu.au). The findings and views reported in this article are those of the author(s) and not those of the original studies or their respective funding agencies.

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